Federation Proceedings

Volume 17 *September 1958* Number 3

FEDERATION OF AMERICAN SOCIETIES FOR EXPERIMENTAL BIOLOGY

FEDERATION BOARD, 1958–1959

HALLOWELL DAVIS, R. F. PITTS, L. N. KATZ, *American Physiological Society*
SEVERO OCHOA, H. G. WOOD, J. T. EDSALL, *American Society of Biological Chemists*
C. D. LEAKE, L. S. GOODMAN, OTTO KRAYER, *American Society for Pharmacology and Experimental Therapeutics*
W. B. WARTMAN, C. C. ERICKSON, E. D. WARNER, *American Society for Experimental Pathology*
W. J. DARBY, D. W. WOOLLEY, R. R. WILLIAMS, *American Institute of Nutrition*
J. E. SMADEL, W. C. BOYD, J. H. DINGLE, *American Association of Immunologists*
J. T. EDSALL, *Chairman,* Harvard University Biological Labs., Cambridge 38, Mass.
M. O. LEE, *Federation Secretary,* 9650 Wisconsin Avenue, Washington 14, D. C.

COMMITTEES

Advisory Committee—J. T. Edsall, Biochemistry, *Chairman;* Otto Krayer, Pharmacology; C. C. Erickson, Pathology; R. R. Williams, Nutrition; J. E. Smadel, Immunology; L. N. Katz, Physiology. *Ex officio:* A. C. Burton and M. O. Lee.

Secretaries Committee—F. W. Putnam, Biochemistry, *Chairman;* H. C. Hodge, Pharmacology; J. F. A. McManus, Pathology; G. M. Briggs, Nutrition; Calderon Howe, Immunology; R. F. Pitts, Physiology.

Public Information Committee—R. W. Engel, Alfred Gilman, R. B. Jennings, Simon Rodbard, E. L. Tatum, B. H. Waksman.

Exhibitors Advisory Committee to the Federation—J. F. Lubben, *Chairman;* Kenneth Ericson, Thomas Lanahan, David Miller, C. R. Roesch, Jr., Melvin Shepherd, Steven K. Herlitz; and Members of the Secretaries Committee.

REPRESENTATIVES TO ORGANIZATIONS

Council of the American Association for the Advancement of Science—K. K. Chen (1960), Orr Reynolds (1959).

American Documentation Institute—M. O. Lee.

Scientific Manpower Commission—M. O. Lee (1959), C. D. Leake (1960).

Future Scientists of America Foundation—R. G. Daggs (1960).

FORMER EXECUTIVE COMMITTEES

Philadelphia, Dec. 28–31, 1913

S. J. Meltzer, *Chairman,* and A. J. Carlson, *Secretary,* Physiology; A. B. Macallum and P. A. Shaffer, Biochemistry; T. Sollmann and J. Auer, Pharmacology.

St. Louis, Dec. 27–30, 1914

G. Lusk, *Chairman,* and P. A. Shaffer, *Secretary,* Biochemistry; T. Sollmann and J. Auer, Pharmacology; R. M. Pearce and G. H. Whipple, Pathology; W. B. Cannon and A. J. Carlson, Physiology.

Boston, Dec. 26–29, 1915

Torald Sollmann, *Chairman,* and John Auer, *Secretary,* Pharmacology; Theobald Smith and Peyton Rous, Pathology; W. B. Cannon and C. W. Greene, Physiology; Walter Jones and P. A. Shaffer, Biochemistry.

New York, Dec. 27–30, 1916

Simon Flexner, *Chairman,* and Peyton Rous, *Secretary,* Pathology; W. B. Cannon and C. W. Greene, Physiology; Walter Jones and Stanley R. Benedict, Biochemistry; Reid Hunt and J. Auer, Pharmacology.

XUM

Minneapolis-Rochester, Dec. 27–29, 1917

Frederic S. Lee, *Chairman,* and Charles W. Greene, *Secretary,* Physiology; Carl L. Alsberg and Stanley R. Benedict, Biochemistry; Reid Hunt and L. G. Rowntree, Pharmacology; Ludvig Hektoen and Howard T. Karsner, Pathology.

Baltimore, April 24–26, 1918

Carl L. Alsberg, *Chairman,* and Stanley R. Benedict, *Secretary,* Biochemistry; Reid Hunt and E. D. Brown, Pharmacology; H. Gideon Wells and Howard T. Karsner, Pathology; Frederic S. Lee and Charles W. Greene, Physiology.

Cincinnati, Dec. 29–31, 1919

A. S. Loevenhart, *Chairman,* and E. D. Brown, *Secretary,* Pharmacology; W. G. MacCallum and Howard T. Karsner, Pathology; Warren P. Lombard and Charles W. Greene, Physiology; Stanley R. Benedict and Victor C. Myers, Biochemistry.

Chicago, Dec. 28–30, 1920

William H. Park, *Chairman,* and Howard T. Karsner, *Secretary,* Pathology; Warren P. Lombard and Charles W. Greene, Physiology; Stanley R. Benedict and Victor C. Myers, Biochemistry; A. S. Loevenhart and Edgar D. Brown, Pharmacology.

New Haven, Dec. 28–30, 1921

J. J. Macleod, *Chairman,* and Charles W. Greene, *Secretary,* Physiology; D. D. Van Slyke and Victor C. Myers, Biochemistry; C. W. Edmunds and Edgar D. Brown, Pharmacology; F. G. Novy and Wade H. Brown, Pathology.

Toronto, Dec. 27–29, 1922

D. D. Van Slyke, *Chairman,* and Victor C. Myers, *Secretary,* Biochemistry; C. W. Edmunds and Edgar D. Brown, Pharmacology; Howard T. Karsner and Wade H. Brown, Pathology; J. J. R. Macleod and Charles W. Greene, Physiology.

St. Louis, Dec. 27–29, 1923

C. W. Edmunds, *Chairman,* and Edgar D. Brown, *Secretary,* Pharmacology; E. L. Opie and Wade H. Brown, Pathology; A. J. Carlson and Charles W. Greene, Physiology; Philip A. Shaffer and Victor C. Myers, Biochemistry.

Washington, Dec. 29–31, 1924

Alfred S. Warthin, *Chairman,* and E. B. Krumbhaar, *Secretary,* Pathology; A. J. Carlson and Walter J. Meek, Physiology; P. A. Shaffer and D. Wright Wilson, Biochemistry; John Auer and E. D. Brown, Pharmacology.

Cleveland, Dec. 28–30, 1925

A. J. Carlson, *Chairman,* and Walter J. Meek, *Secretary,* Physiology; H. C. Sherman and D. Wright Wilson, Biochemistry; John Auer and E. D. Brown, Pharmacology; George H. Whipple and E. B. Krumbhaar, Pathology.

Rochester, N. Y., April 14–16, 1927

E. C. Kendall, *Chairman,* and F. C. Koch, *Secretary,* Biochemistry; John Auer and E. D. Brown, Pharmacology; W. H. Brown and E. B. Krumbhaar, Pathology; J. Erlanger and W. J. Meek, Physiology.

Ann Arbor, April 12–14, 1928

Carl Voegtlin, *Chairman,* and E. D. Brown, *Secretary,* Pharmacology; David Marine and Carl V. Weller, Pathology; Joseph Erlanger and Walter J. Meek, Physiology; E. V. McCollum and D. Wright Wilson, Biochemistry.

Boston, Aug. 19–24, 1929

(The XIIIth International Physiological Congress)

Edward B. Krumbhaar, *Chairman,* and Carl V. Weller, *Secretary,* Pathology; Joseph Erlanger and Walter J. Meek, Physiology; E. V. McCollum and D. Wright Wilson, Biochemistry; Carl Voegtlin and E. D. Brown, Pharmacology.

Chicago, March 26–29, 1930

Walter J. Meek, *Chairman,* and Alfred C. Redfield, *Secretary,* Physiology; W. R. Bloor and Howard B. Lewis, Biochemistry; Carl Voegtlin and E. D. Brown, Pharmacology; William F. Petersen and Carl V. Weller, Pathology.

Montreal, April 8–11, 1931

W. R. Bloor, *Chairman,* and H. B. Lewis, *Secretary,* Biochemistry; George B. Wallace and E. D. Brown, Pharmacology; Frederick L. Gates and C. Phillip Miller, Pathology; Walter J. Meek and Arno S. Luckhardt, Physiology.

Philadelphia, April 27–30, 1932

George B. Wallace, *Chairman,* and V. E. Henderson, *Secretary,* Pharmacology; Samuel R. Haythorn and C. Phillip Miller, Pathology; Walter J. Meek and Arno B. Luckhardt, Physiology; H. C. Bradley and Howard B. Lewis, Biochemistry.

Cincinnati, April 10–12, 1933

Peyton Rous, *Chairman,* and C. Phillip Miller, *Secretary,* Pathology; Arno B. Luckhardt and Frank C. Mann, Physiology; H. C. Bradley and

Howard B. Lewis, Biochemistry; Wm. deB. MacNider and V. E. Henderson, Pharmacology;

New York, March 28–31, 1934

Arno B. Luckhardt, *Chairman,* Frank C. Mann, *Secretary,* and Alexander Forbes, *Treasurer,* Physiology; W. M. Clark and H. A. Mattill, Biochemistry; W. deB. MacNider and V. E. Henderson, Pharmacology; Carl V. Weller and C. Phillip Miller, Pathology.

Detroit, April 10–13, 1935

W. M. Clark, *Chairman,* H. A. Mattill, *Secretary,* and C. H. Fiske, *Treasurer,* Biochemistry; Charles W. Greene and Frank C. Mann, Physiology; R. A. Hatcher and E. M. K. Geiling, Pharmacology; S. Burt Wolbach and Shields Warren, Pathology.

Washington, March 25–28, 1936

V. E. Henderson, *Chairman,* E. M. K. Geiling, *Secretary,* and C. M. Gruber, *Treasurer,* Pharmacology; Frank C. Mann and Andrew C. Ivy, Physiology; H. B. Lewis and H. A. Mattill, Biochemistry; Oskar Klotz and Shields Warren, Pathology.

Memphis, April 21–24, 1937

Alphonse R. Dochez, *Chairman,* and Shields Warren, Pathology; Frank C. Mann and Andrew C. Ivy, Physiology; Howard B. Lewis and H. A. Mattill, Biochemistry; V. E. Henderson and E. M. K. Geiling, Pharmacology. D. R. Hooker, *Secretary.*

Baltimore, March 30–April 2, 1938

William T. Porter, *Honorary President;* Walter E. Garrey, *Chairman,* and Andrew C. Ivy, Physiology; Glenn E. Cullen and H. A. Mattill, Biochemistry; Arthur L. Tatum and G. Philip Grabfield, Pharmacology; C. Phillip Miller and Paul R. Cannon, Pathology. D. R. Hooker, *Secretary.*

Toronto, April 26–29, 1939

Glenn E. Cullen, *Chairman,* and Charles G. King, Biochemistry; Arthur L. Tatum and G. Philip Grabfield, Pharmacology; C. Phillip Miller and Paul R. Cannon, Pathology; Walter E. Garrey and Andrew C. Ivy, Physiology. D. R. Hooker, *Secretary.*

New Orleans, March 13–16, 1940

E. M. K. Geiling, *Chairman,* and G. Philip Grabfield, Pharmacology; Ernest W. Goodpasture and Paul R. Cannon, Pathology; Andrew C. Ivy and Philip Bard, Physiology; William C. Rose and Charles G. King, Biochemistry. D. R. Hooker, *Secretary.*

Chicago, April 15–19, 1941

Shields Warren, *Chairman,* and H. P. Smith, Pathology; Thorne M. Carpenter and L. A. Maynard, Nutrition; Andrew C. Ivy and Philip Bard, Physiology; William C. Rose and Charles G. King, Biochemistry; E. M. K. Geiling and G. Philip Grabfield, Pharmacology. D. R. Hooker, *Secretary.*

Boston, March 31–April 4, 1942

Albert G. Hogan, *Chairman,* and Arthur H. Smith, Nutrition; Philip Bard and Carl J. Wiggers, Physiology; Rudolph J. Anderson and Arnold K. Balls, Biochemistry; E. M. K. Geiling and R. N. Bieter, Pharmacology; Jesse L. Bollman and H. P. Smith, Pathology. Shields Warren, *Ex-Chairman.* D. R. Hooker, *Secretary.*

1943, 1944, 1945: The meetings scheduled for Cleveland were cancelled because of war conditions

Philip Bard, *Chairman,* and W. O. Fenn, Physiology; E. A. Doisy and A. K. Balls, Biochemistry; E. K. Marshall, Jr. and R. N. Bieter, Pharmacology; Balduin Lucké and H. P. Smith, Pathology; L. A. Maynard and A. H. Smith, Nutrition; J. J. Bronfenbrenner and A. F. Coca, Immunology. D. R. Hooker, *Secretary.*

Atlantic City, March 11–15, 1946

Philip Bard, *Chairman;* W. O. Fenn, Physiology; A. B. Hastings and A. K. Balls, Biochemistry; E. E. Nelson and R. N. Bieter, Pharmacology; Balduin Lucké and H. P. Smith, Pathology; W. C. Rose and H. E. Carter, Nutrition; J. J. Bronfenbrenner and A. F. Coca, Immunology. D. R. Hooker, *Secretary.*

Chicago, May 18–22, 1947

A. B. Hastings, *Chairman,* and O. A. Bessey, Biochemistry; M. H. Seevers and H. B. Haag, Pharmacology; P. R. Cannon and F. S. Robscheit-Robbins, Pathology; A. H. Smith and H. E. Carter, Nutrition; Michael Heidelberger and A. F. Coca, Immunology; W. O. Fenn and M. B. Visscher, Physiology. W. H. Chambers, *Secretary.*

Atlantic City, March 15–19, 1948

M. H. Seevers, *Chairman,* and H. B. Haag, Pharmacology; D. H. Sprunt and F. S. Robscheit-Robbins, Pathology; R. M. Bethke and H. E. Carter, Nutrition; L. D. Felton and A. F. Coca, Immunology; W. O. Fenn and M. B. Visscher, Physiology; H. T. Clarke and O. A. Bessey, Biochemistry. W. H. Chambers, *Secretary.*

Detroit, April 18–22, 1949

H. P. Smith, *Chairman,* and F. S. Robscheit-Robbins, Pathology; E. M. Nelson and J. H. Roe, Nutrition; Michael Heidelberger and Jules Freund, Immunology; M. B. Visscher and D. B. Dill, Physiology; H. T. Clarke and O. A. Bessey, Biochemistry; Carl A. Dragstedt and H. B. Haag, Pharmacology. M. O. Lee, *Federation Secretary.*

Atlantic City, April 17–21, 1950

C. G. King, *Chairman,* and J. H. Roe, Nutrition; Thomas Francis, Jr., and Jules Freund, Immunology; C. J. Wiggers and D. B. Dill, Physiology; C. F. Cori and R. W. Jackson, Biochemistry; C. F. Schmidt and H. B. Haag, Pharmacology; J. G. Kidd and S. C. Madden, Pathology. M. O. Lee, *Federation Secretary.*

Cleveland, April 29–May 3, 1951

Geoffrey Edsall, *Chairman,* and Jules Freund, Immunology; D. B. Dill and R. W. Gerard, Physiology; H. B. Vickery and E. H. Stotz, Biochemistry; C. F. Schmidt and H. B. Haag, Pharmacology; J. F. Rinehart and S. C. Madden, Pathology; W. H. Griffith and J. H. Roe, Nutrition. M. O. Lee, *Federation Secretary.*

New York City, April 14–18, 1952

D. B. Dill, *Chairman,* H. E. Essex, R. W. Gerard, Physiology; Vincent du Vigneaud, E. H. Stotz, H. B. Vickery, Biochemistry; McKeen Cattell, C. C. Pfeiffer, C. F. Schmidt, Pharmacology; F. S. Robscheit-Robbins, R. L. Holman, J. F. Rinehart, Pathology; C. M. McCay, J. M. Orten, W. H. Griffith, Nutrition; C. M. MacLeod, J. Y. Sugg, Geoffrey Edsall, Immunology. M. O. Lee, *Federation Secretary.*

FORMER FEDERATION BOARDS

Chicago, April 6–10, 1953

Vincent du Vigneaud, *Chairman;* H. A. Mattill, D. W. Wilson, E. H. Stotz, Biochemistry; K. K. Chen, C. C. Pfeiffer, McKeen Cattell, Pharmacology; S. C. Madden, R. L. Holman, F. S. Robscheit-Robbins, Pathology; P. L. Day, J. M. Orten, C. M. McCay, Nutrition; J. F. Enders, J. Y. Sugg, C. M. MacLeod, Immunology; E. M. Landis, E. F. Adolph, R. W. Gerard, Physiology. M. O. Lee, *Federation Secretary.*

Atlantic City, April 11–16, 1954

K. K. Chen, *Chairman;* H. B. Haag, C. C. Pfeiffer, Pharmacology; D. M. Angevine, C. C. Erickson, S. C. Madden, Pathology; C. A. Elvehjem, J. M. Orten, P. L. Day, Nutrition; T. P. Magill, J. Y. Sugg, J. F. Enders, Immunology; E. F. Adolph, H. E. Essex, E. M. Landis, Physiology; D. W. Wilson, Philip Handler, Vincent du Vigneaud, Biochemistry. M. O. Lee, *Federation Secretary.*

San Francisco, April 11–15, 1955

D. M. Angevine, *Chairman;* R. L. Holman, C. C. Erickson, Pathology; G. R. Cowgill, R. W. Engel, C. A. Elvehjem, Nutrition; A. M. Pappenheimer, Jr., F. S. Cheever, T. P. Magill, Immunology; H. E. Essex, W. F. Hamilton, E. F. Adolph, Physiology; C. G. King, Philip Handler, D. W. Wilson, Biochemistry; C. M. Gruber, C. C. Pfeiffer, H. B. Haag, Pharmacology. M. O. Lee, *Federation Secretary.*

Atlantic City, April 16–20, 1956

G. R. Cowgill, *Chairman,* R. W. Engel, W. H. Sebrell, Jr., Nutrition; Jules Freund, F. S. Cheever, A. M. Pappenheimer, Jr., Immunology; W. F. Hamilton, A. C. Burton, H. E. Essex, Physiology; J. M. Luck, Philip Handler, C. G. King, Biochemistry; E. G. Gross, C. C. Pfeiffer, C. M. Gruber, Pharmacology; H. L. Stewart, C. C. Erickson, R. L. Holman, Pathology. M. O. Lee, *Federation Secretary.*

Chicago, April 15–19, 1957

Jules Freund, *Chairman;* M. W. Chase, F. S. Cheever, Immunology; A. C. Burton, L. N. Katz, W. F. Hamilton, Physiology; H. E. Carter, Philip Handler, J. M. Luck, Biochemistry; Harry Beckman, H. C. Hodge, E. G. Gross, Pharmacology; F. W. Hartman, C. C. Erickson, H. L. Stewart, Pathology; R. R. Williams, R. W. Engel, W. H. Sebrell, Jr., Nutrition. M. O. Lee, *Federation Secretary.*

Philadelphia, April 14–18, 1958

A. C. Burton, *Chairman,* L. N. Katz, Hallowell Davis, Physiology; J. T. Edsall, Philip Handler, H. E. Carter, Biochemistry; Otto Krayer, H. C. Hodge, Harry Beckman, Pharmacology; E. D. Warner, J. F. A. McManus, F. W. Hartman, Pathology; R. R. Williams, G. M. Briggs, R. W. Engel, Nutrition; J. H. Dingle, F. S. Cheever, M. W. Chase, Immunology. M. O. Lee, *Federation Secretary.*

FEDERATION CONSTITUTION AND BYLAWS

Adopted at the New York City Meeting, April 17, 1952

CONSTITUTION

ARTICLE I. *Name*

The name of this organization is the Federation of American Societies for Experimental Biology.

ARTICLE II. *Purpose*

The purposes of the Federation are: to bring together investigators in biological and medical sciences represented by the member Societies; to disseminate information on the results of biological research through publications and scientific meetings; and to serve in other capacities in which the member Societies can function more efficiently as a group than as individual units.

ARTICLE III. *Membership*

SECTION 1. The constituent members of the Federation are, in order of seniority: The American Physiological Society; American Society of Biological Chemists, Inc.; American Society for Pharmacology and Experimental Therapeutics, Inc.; The American Society for Experimental Pathology; American Institute of Nutrition; The American Association of Immunologists.

SECTION 2. Other societies in the field of biological science may apply for membership, and may be admitted by unanimous consent of the Societies which are members at the time of application.

SECTION 3. Any constituent Society may withdraw from the Federation one year following written notification to the Federation Board of its intention.

ARTICLE IV. *Management*

The management of the Federation shall be vested in a Federation Board, on which each of the constituent Societies shall have equal membership. The Chairman of the Federation Board shall be furnished for a one-year term by each Society in its turn as determined by seniority in the Federation and shall be the immediate Past-President of that Society.

ARTICLE V. *Amendments*

SECTION 1. *Amendments.* Amendments to this Constitution may be proposed by any of the constituent Societies. If approved by a two-thirds majority of the Federation Board, the proposal shall then be referred to the constituent Societies. The amendment shall be adopted when approved by all of the constituent Societies.

SECTION 2. *Bylaws.* Bylaws may be adopted or amended by the Federation Board at any meeting of the Board by a vote in which at least two of the representatives from each member Society must concur.

BYLAWS

(Amended, 1958)

ARTICLE I. *The Federation Board*

SECTION 1. The Federation Board shall consist of three representatives from each of the constituent Societies, each of whom shall serve for a term of three years. [Initially, each Society shall designate one of its representatives to serve for one year, another to serve for two years and the third to serve for three years.]

The Federation Board shall have the control and general management of the business affairs of the Federation. The Chairman shall have such powers and duties as are ordinarily exercised by the president of a corporation, including but not limited to the following: a) he shall preside at meetings of the Federation Board; b) he shall sign and execute all contracts, agreements and other legal documents in the name of the Federation upon authorization by the Federation Board.

SECTION 2. The Federation Board shall consider measures of advantage to the Federation. Any constituent Society may refer such measures to the Federation Board for consideration. In the interim between regular meetings of the Federation Board, such measures may be referred to all members of the Board by mail and the members may vote on such measures by mail ballot.

SECTION 3. The Federation Board may take such action, or may authorize the Chairman or the Federation Secretary to take such action, as is approved by at least two representatives of each Society. The actions of the Federation Board shall be reported to the annual business meetings of the member Societies. A quorum of the Federation Board shall consist of not less than twelve Board members, with each constituent Society represented by at least two of its representatives.

SECTION 4. The Federation Board shall appoint annually a Secretary-Treasurer, to be known as the Federation Secretary, who may present matters to the Federation Board for decision but who shall not be entitled to vote.

SECTION 5. The Federation Board shall control all monies of the Federation, determine the annual Federation assessment per member of the constituent Societies, set the subscription rates for Federation publications and the fees of the Placement Service, and appropriate monies for use of its authorized committees and the Federation Secretary. The assessment shall be applied to each Society as of July 1 of each year and shall be due the Federation on that date.

SECTION 6. The Federation Board shall determine times and places of annual meetings of the Federation upon recommendations of the Federation Secretary.

ARTICLE II. *Federation Secretary*

The Federation Secretary shall act as secretary to the Federation Board and be responsible for all Federation records; he shall act as treasurer for the Federation and submit audited financial statements to the Federation Board at the annual meeting; he shall be responsible for making the arrangements for the annual meeting of the Federation; he shall act as managing editor of the publications of the Federation; he shall supervise the Federation's Placement Service and make an annual report on its activity to the Federation Board; and he shall perform such other duties as prescribed by the Federation Board and which do not conflict with the Constitutions and Bylaws of the constituent Societies.

ARTICLE III. *Committees and Representatives*

SECTION 1. The Federation Board shall establish such committees and designate such representatives as desired and receive their reports. The Chairman of the Federation Board shall appoint, with the advice of the Board, the individual committee members and representatives, unless otherwise specified by the Board.

SECTION 2. An Advisory Committee, composed of one member designated annually by each constituent Society from its three representatives to the Federation Board, shall be a standing committee. The Chairman of the Board shall be the designated member from his Society and shall be the Chairman of the Advisory Committee. The Federation Secretary and the immediate Past-Chairman of the Federation Board shall be ex officio members. The Committee shall meet at the call of the Chairman.

SECTION 3. A Public Information Committee shall be a standing committee of the Federation.

SECTION 4. A Secretaries Committee, composed of the Secretaries of the constituent Societies, shall be a standing committee of the Federation. The Chairman of this Committee shall be the Secretary of the senior Society for the year. This Committee shall be responsible for arranging the program of the annual meeting and for setting the registration fee for this meeting, shall act as an editorial board for Federation publications and shall perform such other functions in connection with the annual meeting as prescribed by the Federation Board.

SECTION 5. The Secretaries Committee shall meet during the winter, and a meeting of the entire Federation Board may be associated with this meeting.

ARTICLE IV. *Meetings*

SECTION 1. The Federation shall hold an annual meeting for presentation of papers at the scientific sessions of the constituent Societies or at general sessions of the Federation and for transaction of business.

SECTION 2. Abstracts of papers to be read at an annual meeting of the Federation must be submitted to the Secretary of the Society of which the proposer of the paper is a member, and such proposer must be either an author of the paper or introduce the author(s). No person may present orally more than one paper at the Federation meeting except at the invitation of the Secretary of an individual Society. The oral presentation of any paper shall be by the first named author, except by permission of the Secretary of the Society concerned.

SECTION 3. Each Society shall have the responsibility for selecting and arranging its scientific program and for setting its specific regulations, provided these are not in conflict with the general regulations under Section 2. The authors of the paper may request its transfer to the program of another Society, but this may be done only with the consent of the Secretaries of the two Societies concerned. Secretaries on their own initiative may transfer papers from the program of one Society to that of another. Transfers shall be indicated in the program.

SECTION 4. The abstracts of papers read at scientific sessions of all constituent Societies at the annual meeting shall be published in the FEDERATION PROCEEDINGS. Rules regarding the form and length of the abstracts shall be set by the Secretaries Committee and shall be circulated at least six weeks prior to the date set for receipt of the abstracts.

ARTICLE V. *Publications*

The official publications of the Federation shall be FEDERATION PROCEEDINGS and such other publications as the Federation Board may authorize. FEDERATION PROCEEDINGS shall be owned by the Federation and published under the supervision of the Managing Editor and the Secretaries Committee. One issue, in two parts, shall contain the abstracts of papers

and the program for the annual meeting. One issue shall contain the annual list of the members of the Societies and other pertinent information. Two other issues shall contain papers from the Joint Session, symposia and such other material as may be selected by the Secretaries Committee.

ARTICLE VI. *Placement Service*

The Federation shall maintain a Placement Service the function of which shall be to assist in the placing of professional personnel in suitable positions.

ARTICLE VII. *Seal*

The seal of this Federation shall be circular in form with the name of the Federation around the outer edge and in the center the words, "Incorporated in the District of Columbia January 19, 1954."

PLACEMENT SERVICE

The Federation maintains a service to act as a medium of communication between persons seeking positions in teaching or research and institutions that wish to fill vacancies in these sciences.

The Service does not undertake to recommend or to pass judgment upon applicants. It aims merely to serve as a clearing-house for such information as above stated and to bring into touch with one another candidates for positions and employers. It publishes quarterly lists of applicants in February, May, August and November, semiannual lists of available positions in March and September and a list of fellowships and assistantships in October. Publication of the February list of candidates and the March list of positions precedes the annual meeting, allowing time for correspondence between interested employers and candidates before the dates of the interview services provided them.

Individuals, whether members of the Federation or not, universities, other institutions and organizations desiring to avail themselves of the Service may receive such information as is available.

By action of the Executive Committee the following charges were authorized:

Annual applicant registration fee	$ 3
Annual subscription to all lists of the Service	$20
Single copy of list of candidates	$10
Single copy of available positions	$ 5
Single copy of fellowship list	$ 2

A single copy of the fellowship list will be sent without cost to any member of a constituent Society upon written request.

To be eligible to use the arrangements for interviews at the annual meeting an employer must be an annual subscriber or a subscriber to the then current February list of applicants.

All communications should be addressed to: Federation Placement Service, 9650 Wisconsin Ave., Washington 14, D. C.

FEDERATION BOARD PROCEEDINGS
1957-1958

The Constitution and Bylaws of the Federation are published every year in FEDERATION PROCEEDINGS, but one wonders how many of the members of the constituent Societies have read them, or know of whom the Board consists and what it does. I certainly knew very little of its activities until I found myself a member of the Board, and even after 2 years I found I really did not know the half of it until I became the Chairman.

The Board has 18 members plus one ex-officio, the Federation Secretary. There are 3 representatives of each of the constituent Societies, regardless of the size of the individual Societies. The basic purpose of the Board is cooperation for the mutual good of the Societies and of the disciplines of biological science that they promote. The functions of the Federation are year-round, including the holding of the Spring Federation Meeting. The Board usually meets twice annually. The first meeting is in January, at the time when the Secretaries of the Societies meet for the final coordination of the program for the Annual Meeting, a meeting attended by more than 8,000 scientists. Only one who has had to participate in this can imagine the pace and complexity of this "Operation Federation Meeting," or realize how efficient are Dr. Milton Lee (the Federation Secretary) and his staff, with the Secretaries, in producing order from what might be chaos.

What does the Federation Board talk about all day, and often far into the night? As an example, here are some items, routine and otherwise, on the agenda for the Board meeting in January 1958. As always, the first routine business was concerned with finances and with approving the audited annual statement and the budget for the ensuing year, prepared by Mr. Harvey, the very efficient Business Manager of the Federation. Mr. Harvey's coming to our staff 2 years ago made a very great difference. He was flexible enough to change the format of the usual incomprehensible (to laymen) accountant's balance sheets to one which most of us can understand.

The total income and expenses for 1957 were over $200,000, respectively, which should make members of the Societies realize that the Federation, whether it likes it or not, is almost in big business. Of the total income less than 10% is derived from the assessment on member Societies. The largest items of income are from advertising and exhibits at the Federation's Annual Meetings, and from grants and government contracts with the National Science Foundation and other agencies. For these, the Federation undertakes many operations requiring organization and experience, and acquaintance with the wide field of biological research and its scientists. These projects benefit all the members of individual Societies indirectly by furthering our sciences and their intercommunication. Registration fees at the Annual Meetings are also a large item of income.

The largest item of expense is for salaries, wages, insurance, etc. for the Federation staff. This is about the only item of expense that is not directly self-liquidating. The aim is to keep publications, namely FEDERATION PROCEEDINGS, on a balanced budget. For accounting purposes, the Business Manager has devised a time-cost-accounting system of the expense of running the business office as it is allocated to the various services rendered to different Societies, and to different activities. This system was approved by the Board.

The Federation Secretary is appointed each year (this keeps him on his toes—to continue to please the Board!), and the Board lost no time in unanimously reappointing Dr. Lee for 1958. The details of salary of his staff are then left to him, subject to approval by the proper committee of the Board. This year Mrs. Ann Nixon, who has been serving the Federation so well, was appointed to the new staff post of Administrative Assistant to the Federation Secretary. The appointment of auditors was made and the usual bank resolutions adopted.

The Board agreed that the Federation Placement Service has become one of the most important of Federation services to all members of Societies and to young scientists who, we hope, will eventually become members. Dr. Lee reported on the activities of the Placement Service (1,400 interviews arranged at the 1957 meeting) and the Board discussed how to cope with an operating deficit (about $11,000 in 1957) for this activity. To alleviate this in the fairest way, the Board voted that $1.50 of each registration fee at the Spring Meeting should be earmarked for the Placement Service. Many of us felt that, while the fees for interviews should be kept minimal for applicants and for University employers using the service, there ought to be some way of assessing the commercial laboratories more heavily. However, no satisfactory formula has yet been devised.

The Board heard the Managing Editor's report on FEDERATION PROCEEDINGS. A motion that the section you are now reading, i.e. "Federation Board Proceedings," be created was passed. There was considerable discussion at both the meeting of the Secretaries Committee, which is directly responsible for the abstracts and program issues, and in the Board meeting concerning the mounting costs of reproducing abstracts. Later we learned that an unexpected deficit of approximately $6,000 had occurred in 1957 and this was likely to be increased to $13,000 in 1958. This was due to an increase in the amount of material published and to mounting costs of printing. This necessitated an emergency meeting of the Finance Committee in Philadelphia at the time of the Annual Meeting, to recommend to the Board possible measures of economy. The Finance Committee suggested several minor economies, some of which were adopted by the Board. These were: limiting the listing in the alphabetical directory to 3 lines per member; reproducing the abstracts by offset from authors' copy; printing the directory by offset; and omitting the subject index in the program issue but retaining it in the abstracts issue. Also, authors who do not supply copy in the proper form (there are many) will be charged $5.00 for the alterations that are required. While these economies might save $10,000, they obviously are only stop-gaps. The Board also recommended to the Secretaries Committee, whose prerogative it is, that they increase the registration fee at the Annual Meeting by at least $1.00.

In connection with FEDERATION PROCEEDINGS, there have been some complaints from individuals who belong to more than one Society, regarding dual Federation assessments. Several methods of dealing with this problem were suggested, but most of them were too complicated in bookkeeping, seemed to discriminate between small and large Societies, or carried some other objection. It was recognized by the Board that the Federation assessment was an obligation of a Society, not one to be undertaken at will by any member of a Society. When an individual joins a Society he assumes the existing obligations of that Society. However, to offer some relief to those with multiple memberships, the Board ruled that a member is entitled to a copy of FEDERATION PROCEEDINGS for each membership he holds in the constituent Societies of the Federation. Such additional copies will be sent *upon request.*

Part of the January meeting is always taken up by reports from the Chairman and the Federation Secretary of the many executive decisions and actions that had to be taken in the long interim between the April meeting of the Board and the following January meeting. Representatives to a number of sister organizations have to be appointed, such as American Association for the Advancement of Science, American Documentation Institute, Scientific Manpower Commission and Future Scientists of America. Then there are always a number of ad hoc appointments of persons to represent the Federation at specific meetings, such as centennials of universities, special committees, scientific conferences, Congressional hearings etc. During 1957 the Federation participated in many projects of the National Science Foundation, including the White Mountain High Altitudes Laboratory, Microbiological Facilities, Neurological Congress Awards, Gerontological Congress Awards and the Japanese Symposium on Enzymes. The Federation Office has been asked to administer the financial affairs and act as General Secretariat for the Fifth International Congress on Nutrition to be held in Washington in 1960. The Federation, in cooperation with the National Science Foundation, maintains a Register of Scientific and Technical Personnel in biological sciences which is proving of considerable help in making studies of salaries and distribution of biologists in the United States.

The Federation has been playing an increasingly important role in national policy-making for science, where its influence should be exerted. Dr. Alan T. Waterman, Director of the National Science Foundation, asked the Federation to submit recommendations for appointments to the NSF Board. Since the undersigned is not a U.S. citizen, Dr. Edsall, the 1958-1959 Chairman of the Federation Board, consulted the presidents of the six Societies and the American Institute of Biological Sciences, and a common list of nominees was sent to the National Science Foundation and to the President of the United States.

The Board, through Drs. Lee, Edsall and Katz, sent protests to the U. S. Civil Service Commission about the exclusion of biological scientists from the pay increases recently granted to physical scientists and engineers. As a result, the Commission has now undertaken a survey of salaries paid by non-Federal agencies to biological scientists to evaluate the competitive salary situation. The Board also adopted a resolution on high-school science teacher-education, as requested by the National Academy of Sciences-National Research Council Advisory Board on Education. This urged universities to provide courses in basic concepts in biology and Departments of Education to give credits for these courses towards a Master's Degree in Education.

The Councils of member Societies often refer matters to the Board. Dr. Handler (ASBC) brought up the matter of endorsement of programs for fluoridation of communal water supplies. After considerable discussion of this matter, which has so many controversial aspects, the Board endorsed a resolution, based on the conclusions of the National Research Council, that stated that "in the judgment of this Board the weight of scientific evidence gathered by eminent scientists and scientific

organizations clearly indicates that fluoridation of communal water supplies exerts a beneficial effect in protecting against the development of dental caries and is without demonstrable injurious effects on health." This seemed to us to keep within our competence, and yet be positive in our stand.

At the Annual Meeting in Philadelphia, the Board met on Sunday evening, after an early dinner together, and continued until nearly midnight. There were more interim activities to be reported. Dr. Handler had been appointed representative of the Federation to appear before a Senate Committee in support of the National Institutes of Health's defense of its budget for training programs. Names had been submitted for observers on the coming Symposium on Earth Satellites, organized by the National Academy of Sciences and the U. S. Committee on the International Geophysical Year. Dr. Lee had attended a meeting of the Council on Documentation Research, at Western Reserve University, and reported on this. The Board deliberated on the suggestions of the Finance Committee to meet the deficit in publication of FEDERATION PROCEEDINGS, as already outlined. Much of the time was occupied by a discussion of the Public Information Committee of the Federation and on what we should do about public relations. On this topic there was much divergence of opinion. Most of the Board realized the importance of public relations and an efficient organization to handle these, especially at the Federation Meetings where the Public Information Committee, composed of devoted volunteers under Dr. Richard Winzler as Chairman, has for many years assisted in the operation of a press room. However, this has become too onerous a task for volunteers. Some feel we need a year-round activity in this field, others are reluctant to see the Federation go far into this sort of venture. The Federation has always been doubtful whether we could represent all of biological science in such an endeavor, and so when it appeared possible to work jointly with the American Institute of Biological Sciences on establishing an organizing committee, we felt it was a solution. However, Dr. Fenn, President of AIBS, reported to the Board that his organization has not yet agreed on the joint procedure suggested. After many proposals to initiate a separate organization for the Federation, or to set up two coordinated and cooperating offices, one for FASEB and one for AIBS, had been offered but not adopted, the Board reluctantly left the matter in abeyance, knowing that sooner or later this problem must be faced and a solution found.

The Chairman had appointed an ad hoc committee (Dr. Handler, Chairman; Dr. Briggs and Dr. Cheever) to consider criteria for the admission of new Societies to the Federation. Obviously we must consider this matter or remain static in the face of development in biological science. This committee reported briefly but was asked to give a further report at the meeting next January. Dr. Chauncey Leake reported on a meeting of the Scientific Manpower Commission which he had attended as the representative of the Federation.

Dr. Katz reported that Professor Houssay, President of the XXI International Congress of Physiological Sciences, had asked the American Physiological Society to mail announcements of the Congress to all interested persons in North America. The Board approved the mailing of this information by the Federation to every member of the constituent Societies. The Board also made the income of a trust fund, in the care of the Federation, available for travel grants to this Congress (in Buenos Aires in 1959) and named the National Committee of IUPS to select the travel grant awardees.

Every year the Board has a new Chairman, by rotation among the Past-Presidents of the six Societies, and each new Chairman probably has some chief goal to aim for in his brief term of office. The aim of your immediate Past-Chairman was to stir up the members of the Board to consider whether or not the present Constitution and operating procedures of the Board, as outlined by the Bylaws, were best suited to efficient functioning, for the benefit of all our constituents, in the conditions of 1957-1958 and years to come. To this end, early in the Fall of 1957 he sent to all members of the Board and to several Past-Chairmen and "elder statesmen" letters designed to be provocative, and suggested that perhaps the Board was too large to be efficient, had too little provision for continuity and the benefit of experience on the Board, that the veto power (2 members of any one Society can veto a measure desired by all others on the Board) was perhaps illogical and unnecessary, that the Board ought to have an authorized executive committee to help its Chairman make the many urgent policy decisions in the interim between meetings, and so on. The letters achieved the "provocation." Replies reflected a remarkable variety, and even conflict, of opinion on many points, but all courteously accepted the effort at self-analysis as useful. At the January meeting the Board was eager to hold discussions on the whole matter, but pressure of necessary business made the time available very short. Between then and the April meetings, the Chairman circulated a summary of opinions gathered from the many replies to his letters, most of them very thoughtful and stimulating. Rather to our astonishment, it appeared that there was a general recognition that the organization could be improved and considerable areas of agreement on which action could and should be taken. There was a lack of time for discussion, in the brief attempt to do so at the first meeting of the Board in Philadelphia, which resulted only in a number of conflicting motions, each going down to defeat. The Board decided to hold an extra meeting at lunch time between the scientific

sessions, and this time there was a unanimity which astonished us all. Several motions were passed that should have far-reaching effects on the efficiency of operation of the Board. In my opinion, the great advance is the finding of a formula of representation by Societies that will ensure continuity of experience on the Board of all its members, where before the different systems in different Societies made great gaps in continuity possible. For example, it was possible, and had happened, that a man found himself Chairman of the Board after only a very brief experience on the Board. This new formula is acceptable to all Societies because, while ensuring the principle of continuity, it yet preserves complete autonomy within Societies, as there should be. The second great step forward is that the new Bylaws give the Chairman the moral support of an Advisory Committee, much smaller than the 18-man Board, yet representative of all the Societies, which will help the Chairman make the many decisions he must make throughout the year. The changes in the Bylaws are given below. The Board does not regard them as final but rather as experimental, which must be tried before we find the best solution to the problem of organization. Once we have started to accept the possibility of improvement, it may well be that full solution, even of the vexing problem of the veto, will come more easily than anyone has believed possible.

Here are the pertinent parts of the Minutes of the Board meeting which include the changes in the Bylaws passed with such unanimity at the "historic" meeting on April 16, 1958.

"First paragraph of Section 1, Article I, shall be replaced with the following:

'The Federation Board shall consist of three representatives from each of the constituent Societies, each of whom shall serve for a term of three years. [Initially, each Society shall designate one of its representatives to serve for one year, another to serve for two years and the third to serve for three years.]'"

"Article III, Section 2, shall read:

'An Advisory Committee, composed of one member designated annually by each constituent Society from its three representatives to the Federation Board, shall be a standing committee. The Chairman of the Board shall be the designated member from his Society and shall be the Chairman of the Advisory Committee. The Federation Secretary and the immediate Past-Chairman of the Board shall be ex-officio members. The Committee shall meet at the call of the Chairman.'

"Present Sections 2, 3 and 4 should be designated Sections 3, 4 and 5, respectively."

"... a Motion that the Headquarters Committee be discharged as of July 1, 1958 and that the Advisory Committee assume the duties of the Headquarters Committee. Motion was ... carried unanimously."

"... a Motion that the Finance Committee also be abolished as of July 1, 1958 and that its functions be assumed by the Advisory Committee. Motion was ... carried unanimously."

Each successive Chairman must feel that his year was unique, but certainly there are features of 1957-1958 for the Federation that were remarkable. The services of the Federation to the Societies and to biological science have considerably increased. The attendance at the Federation's Annual Meeting in April broke all records with a total registration of 9,136. There were more inter-Society sessions (fulfilling the real purpose of the Federation, namely cross-fertilization of ideas) than ever before. The Board, in an extraordinary meeting, passed several important amendments to its Bylaws without a dissenting vote, and this exhibited a spirit of cooperation for the common good that was most gratifying.

As retiring Chairman, I take this opportunity to thank all the members of the Board for their cooperation, especially for their patience and broad-mindedness in discussing many opinions on which they initially differed so strongly, and for their great forbearance with their Chairman. Dr. Milton Lee, Miss Sara Leslie, his able assistant who has helped guide the Federation for many years, and their staff, especially our efficient Minute-taker, Mrs. Ann Nixon, were towers of strength.

Following the example of Captain Hook in "Peter Pan," as I come to my end as Chairman, I cry, "Floreat FASEB."

Alan C. Burton

Federation of American Societies for Experimental Biology

Exhibit A. Balance Sheet—December 31, 1957

ASSETS:			
Cash in banks			$ 29,606
Accounts receivable			27,228
Building and loan deposits			16,158
Supplies inventory			3,280
Prepaid expenses and deposits			17,787
Fixed Assets:	*Cost*	*Reserves for Depreciation*	
Land	$ 25,823	$ -0-	25,823
Buildings and improvements	125,131	11,446	113,685
Furniture and equipment	33,586	11,625	21,961
Trust fund investments at cost (market value $25,800)			17,492
Total Assets			$273,020
DEDUCT—LIABILITIES:			
Accounts payable and accrued expenses			$ 27,192
Note payable to the Riggs National Bank—4½% (secured by pledge of land and building; payable $2,250 semi-annually)			46,500
Deferred income			98,114
Total Liabilities			$171,806
NET ASSETS, REPRESENTING FUND CAPITAL (PER EXHIBIT B)			$101,214

Exhibit B. Statement of Income and Expenses and Fund Capital for Year Ended December 31, 1957

INCOME:	
FEDERATION PROCEEDINGS:	
Assessments on member societies	$ 19,325
Subscriptions, advertising, reprints, etc.	64,270
Annual meeting	90,271
Placement information service	6,952
Grants and Government contracts	45,906
Rental income	4,666
Dividends and other income	1,917
Total Income	$233,307
EXPENSES:	
Salaries, wages, Social Security, group insurance, and pension	$109,563
Printing and engraving	40,942
Advertising and exhibit commissions	19,244
Projection and public address services	4,493
Professional and other personal services	8,175
Travel awards to Japanese Enzyme Symposium	22,000
Rentals of meeting rooms, exhibit halls and equipment	3,794
Supplies and duplicating (less duplicating services charged to other activities $7,742)	2,960
Communications, shipping and travel	18,149
Operation and maintenance of buildings, grounds, and equipment	20,067
Other expenses and supplies	4,385
	$253,772
Less: Portion of business services paid by American Physiological Society	−29,919
Total Expenses	$223,853
EXCESS OF INCOME OVER EXPENSES	$ 9,454
FUND CAPITAL:	
Balance, December 31, 1956	91,760
Balance, December 31, 1957	$101,214

THE AMERICAN PHYSIOLOGICAL SOCIETY

Founded December 30, 1887; Incorporated June 2, 1923

OFFICERS, 1958–1959

President—Hallowell Davis, Central Institute for the Deaf, St. Louis, Missouri.

President-Elect (Secretary)—R. F. Pitts, Cornell University Medical College, New York City.

Past-President—L. N. Katz, Michael Reese Hospital, Chicago, Illinois.

Council—Hallowell Davis, R. F. Pitts, L. N. Katz, T. C. Ruch (1959), James Daniel Hardy (1960), H. S. Mayerson (1961), J. H. Comroe, Jr. (1962).

Executive Secretary-Treasurer—R. G. Daggs, 9650 Wisconsin Avenue, Washington 14, D. C.

Administrative Advisor—M. O. Lee, 9650 Wisconsin Avenue, Washington 14, D. C.

STANDING COMMITTEES

Membership Advisory—T. C. Ruch (1959), *Chairman;* and Members of the Council.

Educational Matters—H. W. Davenport (1960), *Chairman;* C. L. Prosser (1959), E. F. Adolph (1961), A. W. Martin, Jr. (1961), Hermann Rahn (1962).

Use and Care of Animals—R. F. Pitts (1959), *Chairman;* H. W. Davenport (1959), A. B. Otis (1961).

Placement of Senior Physiologists—D. B. Dill (1962), *Chairman;* W. O. Fenn (1961), W. F. Hamilton (1960), E. M. Landis (1962).

Porter Fellowship—R. F. Pitts (1959), *Chairman;* Hermann Rahn (1960), J. M. Brookhart (1961).

Program Advisory—J. H. Comroe, Jr. (1960), *Chairman.*

Committee on International Physiology of the American Physiological Society—W. O. Fenn (1959), *Chairman;* R. W. Gerard (1961), M. B. Visscher (1963).

Historian—John Field, II.

REPRESENTATIVES TO OTHER ORGANIZATIONS

American Association for the Advancement of Science—F. A. Hitchcock (1960).

American Documentation Institute—M. O. Lee (1961).

American Institute of Biological Sciences—W. O. Fenn (1959).

National Research Council—Division of Biology and Agriculture, J. O. Hutchens (1961); Division of Medical Sciences, R. W. Gerard (1961).

Council on Medical Education and Hospitals, A.M.A.—J. S. Gray (1961).

A.P.S. Members of the United States National Committee for the International Union of Physiological Sciences—W. O. Fenn (1959), R. W. Gerard (1961), M. B. Visscher (1963), E. M. Landis (alternate).

Council for Coordination of International Congresses—M. B. Visscher.

Council of International Organizations in Medical Sciences—M. B. Visscher.

A.P.S. Representatives on Joint Symposium Committee on Neuropharmacology—H. E. Himwich (1961), T. C. Ruch (1961).

EDITORIAL BOARDS

Board of Publication Trustees—M. B. Visscher (1959), *Chairman;* W. F. Hamilton (1960), Philip Bard (1961).

Managing Editor—M. O. Lee.

Associate Editor, Physiological Reviews—R. G. Daggs.

American Journal of Physiology and Journal of Applied Physiology—W. D. Armstrong (1960), R. S. Alexander (1961), E. J. Baldes (1959), S. B. Barker (1961), R. W. Berliner (1959), A. F. Cournand (1959), R. C. de Bodo (1960), Philip Dow (1960), W. S. Fowler (1959), Robert Galambos (1959), Robert Gaunt (1959), A. S. Gilson, Jr. (1960), H. D. Green (1960), E. G. Gross (1959), A. C. Guyton (1961), H. K. Hartline (1959), A. B. Hastings (1959), H. E. Himwich (1959), Alexander Hollaender (1960), L. B. Jaques (1961), F. H. Johnson (1961), R. E. Johnson (1961), Ancel Keys (1960), W. J. Kolff (1961), Nathan Lifson (1960), G. K. Moe (1959), W. F. H. M. Mommaerts (1960), H. M. Patt (1960), Hermann Rahn (1960), R. L. Riley (1959), Sidney Roberts (1959), Jane Russell (1960), P. F. Salisbury (1961), S. J. Sarnoff (1961), O. H. Schmitt (1960), W. H. Seegers (1959), E. E. Selkurt (1960), H. L. White (1960), E. H. Wood (1960), W. B. Youmans (1960). *Consultant Editors:* A. Albert (1960), A. C. Burton (1960), J. H. Comroe, Jr. (1961), W. O. Fenn (1959), Alfred Gilman (1959), A. C. Ivy (1960), L. N. Katz (1959), C. N. H. Long (1960), R. F. Pitts (1960), J. P. Quigley (1960) T. C. Ruch (1961).

Physiological Reviews—H. S. Mayerson (1961), *Chairman;* C. F. Code (1960), H. J. Curtis (1959), E. M. Landis (1960), V. B. Mountcastle (1960), A. B. Otis (1961), I. H. Page (1960), R. M. Reinecke (1961). *Appointees of the Society of General Physiologists*—K. V.

Thimann (1959), D. R. Griffin (1960); *Appointees of the Society of Biological Chemists*—J. M. Buchanan (1960), E. L. Smith (1960). *European Editorial Committee:* G. Kahlson (1959), *Chairman;* H. Barcroft (1959), A. Fessard (1959), E. Lundsgaard (1959), R. H. S. Thompson (1959).

Annual Review of Physiology—John Field, II, (1960), *Chairman;* J. M. Brookhart (1960), H. W. Davenport (1960), G. K. Moe (1960).

PAST OFFICERS

Presidents—**1888** H. P. Bowditch. **1889–1890** S. W. Mitchell. **1891–1895** H. P. Bowditch. **1896–1904** R. H. Chittenden. **1905–1910** W. H. Howell. **1911–1913** S. J. Meltzer. **1914–1916** W. B. Cannon. **1917–1918** F. S. Lee. **1919–1920** W. P. Lombard. **1921–1922** J. J. R. MacLeod. **1923–1925** A. J. Carlson. **1926–1929** J. Erlanger. **1930–1932** W. J. Meek. **1933–1934** A. B. Luckhardt. **1935** C. W. Greene. **1936–1937** F. C. Mann. **1938–1939** W. E. Garrey. **1938** W. T. Porter, *Honorary President.* **1940–1941** A. C. Ivy. **1942–1946** P. Bard. **1947** W. O. Fenn. **1948** M. B. Visscher. **1949** C. J. Wiggers. **1950** H. C. Bazett (April to July); D. B. Dill. **1951** R. W. Gerard. **1952** E. M. Landis. **1953** E. F. Adolph. **1954** H. E. Essex. **1955** W. F. Hamilton. **1956** A. C. Burton. **1957** L. N. Katz.

Secretaries—**1888–1892** H. N. Martin. **1893–1894** W. P. Lombard. **1895–1903** F. S. Lee. **1904** W. T. Porter. **1905–1907** L. B. Mendel. **1908–1909** R. Hunt. **1910–1914** A. J. Carlson. **1915–1923** C. W. Greene. **1924–1929** W. J. Meek. **1930** A. C. Redfield. **1931–1932** A. B. Luckhardt. **1933–1935** F. C. Mann. **1936–1939** A. C. Ivy. **1940–1941** Philip Bard. **1942** C. J. Wiggers. **1943–1946** W. O. Fenn. **1947** M. B. Visscher.

Treasurers—**1888–1892** H. N. Martin. **1893–1894** W. P. Lombard. **1895–1903** F. S. Lee. **1904** W. T. Porter, **1905–1912** W. B. Cannon. **1913–1923** J. Erlanger. **1924–1926** C. K. Drinker. **1927–1936** Alexander Forbes. **1937–1940** W. O. Fenn. **1941** C. J. Wiggers. **1942–1946** Hallowell Davis. **1947** D. B. Dill.

Executive Secretary-Treasurer—**1948–1956** M. O. Lee. **1956** R. G. Daggs.

CONSTITUTION AND BYLAWS

Adopted at the 1953 Spring Meeting

CONSTITUTION

ARTICLE I. *Name*

The name of this organization is THE AMERICAN PHYSIOLOGICAL SOCIETY.

ARTICLE II. *Purpose*

The purpose of the Society is to promote the increase of physiological knowledge and its utilization.

BYLAWS

(Amended April 1958)

ARTICLE I. *Membership*

SECTION 1. The Society shall consist of members, honorary members and associate members.

SECTION 2. *Members.* Any person who has conducted and published meritorious original research in physiology and/or biophysics and who is a resident of North America shall be eligible for membership in the Society.

SECTION 3. *Honorary Members.* Distinguished scientists of any country who have contributed to the advance of physiology shall be eligible for proposal as honorary members of the Society.

SECTION 4. *Associate Members.* Advanced graduate students in physiology at a predoctoral level, teachers of physiology, and investigators who have not yet had the opportunity or time to satisfy the requirements for full membership shall be eligible for associate membership in the Society provided they are residents of North America.

ARTICLE II. *Officers*

SECTION 1. The management of the Society shall be vested in a Council consisting of the President, the President-Elect, the Past-President for the previous year, and four other members. The terms of the President and of the President-Elect shall be one year. The terms of the four additional Councilors shall be four years each and they shall not be eligible for immediate reelection except those who have served for two years or less in filling interim vacancies. A person may serve only one term as President, except that if the President-Elect becomes President after September 30 he shall continue as President for the year beginning the next July 1st.

SECTION 2. Nomination and election of a President-Elect and Councilor(s) shall be by ballot at the Spring meeting of the Society. They shall assume office on July 1 following their election.

SECTION 3. The President-Elect shall serve as Vice-President and Secretary. Should he have to function as President prematurely, the Council shall select from among its own members a Secretary.

SECTION 4. The Council shall be empowered to appoint and compensate an Executive Secretary-Treasurer who shall assist it in carrying on the functions of the Society, including the receipt and disbursement of funds under the direction of the Council.

SECTION 5. The Council may fill any interim vacancies in its membership or vacancies on any Board or Committee of the Society, unless otherwise provided.

ARTICLE III. *Dues*

SECTION 1. The annual assessment on members and on associate members shall be determined by the Council and shall be due in advance on July 1.

SECTION 2. A member whose dues are two years in arrears shall cease to be a member of the Society, unless after payment of his dues in arrears and application to the Council, he shall be reinstated at the next Spring meeting by special vote of the Council. It shall be the duty of the Secretary to notify the delinquent of his right to request reinstatement.

SECTION 3. A member who has retired from employment because of illness or age may, upon application to the Council, be relieved from the payment of the annual member assessment.

ARTICLE IV. *Meetings*

SECTION 1. A meeting of the Society for transacting business, electing officers and members, presenting communications, and related activities, shall be held in the Spring of each year, with other member Societies of the Federation of American Societies for Experimental Biology, except that under exceptional circumstances the Council may cancel such a meeting.

SECTION 2. A Fall meeting of the Society shall be held at a time and place determined by the Council, for presenting communications and for transacting business except the election of officers.

SECTION 3. Special meetings of the Society or of the Council may be held at such times and places as the Council may determine.

SECTION 4. Regional meetings of the Society, for the purpose of presenting scientific communications, may be authorized by the Council.

ARTICLE V. *Publications*

SECTION 1. The official organs of the Society shall be the American Journal of Physiology, the Journal of Applied Physiology, Physiological Reviews and such other publications as the Society may own.

SECTION 2. A Board of Publication Trustees, composed of three members of the Society and appointed by the Council, shall be vested with with the full power of the Society to control and manage, both editorially and financially, all of the publications of the Society; to appoint editorial boards; to appoint and compensate a Managing Editor; and to control all publication funds, none of which, however, may be diverted from support of the publications of the Society except by consent of the Council. The term of each member of the Board shall be three years; a member may not serve more than two consecutive terms. The Council shall designate the Chairman of the Board, and shall receive an annual report on the finances, publications and policies. A summary of this report shall be presented to the Society at the Spring meeting.

The Chairman of the Board of Publication Trustees shall be a member ex-officio of the Council but shall have no vote.

ARTICLE VI. *Committees and Representatives*

The Council may appoint such special and standing committees as it deems necessary or that are voted by the Society.

The Council may name members of the Society as representatives to other organizations whenever it deems such action desirable.

ARTICLE VII. *Standing Rules*

1. *Election to Membership.* Two members of the Society must join in proposing a person for membership, in writing and with a statement of his qualifications. The Council may, from the persons so proposed, nominate candidates for election to membership. Nominations shall be presented at Spring and Fall meetings; a two-thirds majority vote of the members present and voting at the next following Fall or Spring meeting shall be necessary for election.

If a Spring or Fall meeting of the Society is not held, the procedures of nomination and/or election of new members may be effected by mail.

The names of the candidates nominated by the Council for membership and statements of their qualifications signed by their proposers shall be available for inspection by members during the Society meetings at which their election is considered.

2. *Election to Honorary Membership.* The proposal of an honorary member shall be made by two members of the Society to the Council in writing. The Council may, from the candidates so proposed, make nominations to the Society at a Spring meeting. A two-thirds majority vote of the members present shall be necessary for election.

Honorary members shall have the privilege of attending business sessions of the Society but shall have no vote. They shall pay no membership fees.

3. *Election to Associate Membership.* Associate members shall be proposed, nominated and elected in the same manner as full members.

Associate members shall have the privilege of attending business sessions of the Society but shall have no vote. Associate members may be nominated for full membership.

4. *Presentation of Papers.* At a Spring meeting of the Society, held in conjunction with the Federation meetings, a member or honorary member may present orally or by title, be co-author of, or introduce not more than one scientific paper, except upon invitation of the Council. An associate member or a non-member may present orally one scientific paper only if sponsored by a full member of the Society. At a

Fall meeting, a member, honorary member, or associate member may present orally not more than one paper, except upon invitation of the Council.

Upon invitation by the Council, a member may contribute papers to specially designated sessions of the Society without forfeit of his privilege of presenting a regular scientific communication.

5. There shall be a Committee on Membership appointed by and advisory to the Council.

6. There shall be a Program Advisory Committee appointed by the Council.

ARTICLE VIII. *General*

SECTION 1. *Amendments.* These Bylaws, except Article VII, may be amended at any Spring meeting of the Society by a three-fourths majority vote of the members present.

The Standing Rules of Article VII may be amended by a majority vote of the members present at either a Spring or Fall meeting of the Society.

SECTION 2. *Quorum.* At all business meetings of the Society fifty members shall constitute a quorum.

SECTION 3. *Parliamentary Authority.* The rules contained in Roberts Rules of Order shall govern the conduct of the business meetings of the Society in all cases to which they are applicable and in which they are not inconsistent with the Bylaws or special rules of order of the Society.

AMERICAN SOCIETY OF BIOLOGICAL CHEMISTS

Founded December 6, 1906; Incorporated September 12, 1919

OFFICERS, 1958–1959

President–Severo Ochoa, New York University, New York City.

President-Elect–H. G. Wood, Western Reserve University, Cleveland, Ohio.

Past-President–J. T. Edsall, Harvard University, Cambridge, Massachusetts.

Secretary–F. W. Putnam, University of Florida, Gainesville.

Treasurer–Stanford Moore, Rockefeller Institute for Medical Research, New York City.

Council–Severo Ochoa, H. G. Wood, J. T. Edsall, F. W. Putnam, Stanford Moore, W. D. McElroy (1956–59), H. A. Lardy (1957–60), Philip Handler (1958–61).

STANDING COMMITTEES

Nominating Committee–Fritz Lipmann (1958–59), *Chairman;* C. B. Anfinsen, Jr. (1957–59), P. D. Boyer (1957–59), J. M. Buchanan (1957–59), J. S. Fruton (1958–59), I. C. Gunsalus (1958–59), Arthur Kornberg (1958–59), Hans Neurath (1957–59), E. L. Smith (1958–59).

Editorial Committee–W. H. Stein (1955–61), *Chairman;* C. A. Elvehjem (1953–59), V. H. Cheldelin (1953–59), R. Y. Stanier (1957–59), Arthur Kornberg (1955–61), H. G. Wood (1955–61), B. L. Horecker (1957–63), I. C. Gunsalus (1957–63), Birgit Vennesland (1957–63).

Finance Committee–P. P. Cohen (1951–56, 1956–61), *Chairman;* J. T. Edsall (1957–63), Severo Ochoa (1958–59), W. H. Stein (1958–59), Stanford Moore (1956–59).

Historian–H. T. Clarke.

Legal Counsel–A. S. Davis.

Editorial Board, Journal of Biological Chemistry

J. T. Edsall (1954–63), *Editor;* R. A. Alberty (1958–63), E. G. Ball (1950–55, 1955–60), Konrad Bloch (1951–56, 1956–61), G. B. Brown (1954–59), H. E. Carter (1950–55, 1955–60), H. N. Christensen (1958–63), H. T. Clarke (1956–61), M. Cohn (1958–63), W. E. Cohn (1955–60), C. F. Cori (1941–), E. A. Doisy (1938–), W. H. Griffith (1949–54, 1954–59), A. B. Hastings (1941–54, 1954–59), Leslie Hellerman (1951–56, 1956–61), E. P. Kennedy (1958–63), H. A. Lardy (1958–63), A. L. Lehninger (1954–59), Alton Meister (1958–63), Stanford Moore (1950–55, 1955–60), Hans Neurath (1954–59), Severo Ochoa, (1950–55, 1955–60), E. L. Smith (1958–63), E. E. Snell (1949–59), W. M. Sperry (1949–59), DeWitt Stetten, Jr. (1954–59), H. B. Vickery (1941–), Oskar Wintersteiner (1951–56, 1956–61).

SPECIAL COMMITTEES

Membership Committee–B. L. Horecker (1956–59), *Chairman;* M. J. Coon (1956–59), Alton Meister (1957–60), V. R. Potter (1958–61), G. W. Schwert (1957–60), E. R. Stadtman (1958–61).

Program Committee–Philip Handler, Henry Kamin, G. W. Schwert, Frederick Bernheim.

Revision of Bylaws–H. B. Vickery, *Chairman;* Vincent du Vigneaud, A. B. Hastings.

Clinical Chemistry, 1954–59–Robert Hill, *Chairman;* R. M. Archibald, M. F. Mason, W. J. Darby, A. J. Quick.

Committee on Nomenclature (Joint with American Institute of Nutrition)–E. E. Snell, W. M. Sperry.

Travel Awards–A. L. Lehninger (1959), *Chairman;* I. C. Gunsalus (1960), DeWitt Stetten, Jr. (1961), E. E. Snell (1962), E. P. Kennedy (1963).

REPRESENTATIVES TO OTHER ORGANIZATIONS

American Association for the Advancement of Science–W. F. Loomis (1958–60), Abraham White (1958–60).

National Committee on Biochemistry–Severo Ochoa (1956–59), C. A. Elvehjem (1957–60), E. L. Smith (1958–61).

National Research Council–Division of Biology and Agriculture, F. E. Deatherage (1958–61); Division of Chemistry and Chemical Technology, Sidney Weinhouse (1958–61); Division of Medical Sciences, W. W. Westerfeld (1956–59); Board of International Critical Tables, W. M. Clark; Committee on Biological Chemistry, Division of Chemistry and Chemical Technology, W. D. McElroy (1956–59), G. B. Brown (1957–60), H. G. Wood (1958–61).

Committee on Organic Nomenclature, A.C.S.–H. S. Loring.

American Board of Clinical Chemistry, Inc.–W. M. Sperry, R. M. Archibald, J. H. Roe.

PAST OFFICERS

Presidents–**1907** R. H. Chittenden. **1908** J. J. Abel. **1909** Otto Folin. **1910** T. B. Osborne. **1911** L. B. Mendel. **1912–1913** A. B. Macallum. **1914** Graham Lusk. **1915–1916** Walter Jones. **1917–1918** C. L. Alsberg. **1919–1920** S. R.

Benedict. 1921–1922 D. D. Van Slyke. 1923–1924 P. A. Shaffer. 1925 H. C. Sherman. 1926 E. C. Kendall. 1927–1928 E. V. McCollum. 1929–1930 W. R. Bloor. 1931–1932 H. C. Bradley. 1933–1934 W. M. Clark. 1935–1936 H. B. Lewis. 1937–1938 G. E. Cullen. 1939–1940 W. C. Rose. 1941–1942 R. J. Anderson. 1943–1944 E. A. Doisy. 1945–1946 A. B. Hastings. 1947–1948 H. T. Clarke. 1949 C. F. Cori. 1950 H. B. Vickery. 1951 V. du Vigneaud. 1952 H. A. Mattill. 1953 D. W. Wilson. 1954 C. G. King. 1955 J. M. Luck. 1956 H. E. Carter. 1957 J. T. Edsall.

Vice-Presidents–1907 J. J. Abel. 1908 Otto Folin. 1909 T. B. Osborne. 1910 L. B. Mendel. 1911 A. B. Macallum. 1912–1913 Graham Lusk. 1914–1915 C. L. Alsberg. 1916 F. P. Underhill. 1917–1918 A. P. Mathews. 1919–1920 D. D. Van Slyke. 1921–1922 P. A. Shaffer. 1923–1924 H. C. Sherman. 1925 E. C. Kendall. 1926 E. V. McCollum. 1927–1928 W. R. Bloor. 1929–1930 H. C. Bradley. 1931–1932 W. M. Clark. 1933–1934 H. B. Lewis. 1935–1936 G. E. Cullen. 1937–1938 W. C. Rose. 1939–1940 R. J. Anderson. 1941–1942 E. A. Doisy. 1943–1944 A. B. Hastings. 1945–1946 H. T. Clarke. 1947–1948 C. F. Cori. 1949 H. B. Vickery. 1950 V. du Vigneaud. 1951 H. A. Mattill. 1952 D. W. Wilson. 1953 C. G. King. 1954 J. M. Luck.

Presidents-Elect–1955 H. E. Carter. 1956 J. T. Edsall. 1957 S. Ochoa.

Secretaries–1907–1909 W. J. Gies. 1910–1912 A. N. Richards. 1913–1915 P. M. Shaffer. 1916–1918 S. R. Benedict. 1919–1923 V. C. Myers. 1924–1925 D. W. Wilson. 1926 F. C. Koch. 1927–1928 D. W. Wilson. 1929–1932 H. B. Lewis. 1933–1937 H. A. Mattill. 1938–1940 C. G. King. 1941–1945 A. K. Balls. 1946–1948 O. A. Bessey. 1949 R. W. Jackson. 1950–1953 E. H. Stotz. 1953–1958 P. Handler.

Treasurers–1907–1909 L. B. Mendel. 1910–1912 Walter Jones. 1913–1916 D.D. Van Slyke. 1917–1921 H. C. Bradley. 1922–1925 W. R. Bloor. 1926–1930 G. E. Cullen. 1931–1935 C. H. Fiske. 1936–1940 A. B. Hastings. 1941–1945 W. C. Stadie. 1946–1950 E. A. Evans, Jr. 1951–1956 P. P. Cohen. 1957 Stanford Moore.

CONSTITUTION

From the Articles of Incorporation

1. The name of the proposed corporation is "AMERICAN SOCIETY OF BIOLOGICAL CHEMISTS, INCORPORATED."

2. The purposes for which this corporation is formed are to further the extension of biochemical knowledge and to facilitate personal intercourse between American investigators in biological chemistry.

BYLAWS

(As revised, 1954)

ARTICLE I. *Membership*

SECTION 1. *Eligibility for Membership.* Qualified investigators who have conducted and published meritorious original investigations in biological chemistry shall be eligible for membership in the Society.

SECTION 2. *Nomination.* Nominations for membership shall be made and seconded by members of the Society on blanks furnished by the Secretary. Nominations shall be submitted to the Council who shall determine eligibility and make recommendation to the Society at a regular meeting.

SECTION 3. *Election to Membership.* A nominee for membership may be voted for by ballot at any meeting of the Society after the Council has declared the nominee to be eligible and endorsed. A majority of the ballots cast shall elect.

SECTION 4. *Forfeiture.* A. Any member who grants the use of his name for the advertisement or for the promotion of the sale of a product to be used for medicine or for food, this product not being one of demonstrated usefulness or value, or who shall act in a manner that is not in the public interest, shall forfeit his membership.

B. The Council shall have authority to announce forfeiture of membership provided that a copy of the charges, together with a written notice of a hearing thereon with time and place specified, shall have been delivered at least thirty days before the date of such hearing to the member charged with the violation.

SECTION 5. *Expulsion.* Upon recommendation of the Council any member may be expelled by a majority vote of the members present at a Business Meeting of the Society, provided that a copy of the charges together with a written notice of a hearing with time and place specified, shall have been delivered to the member at least thirty days before the date of such hearing.

ARTICLE II. *Meetings and Quorum*

SECTION 1. *Annual.* The annual meeting of the Society shall be held on the date fixed by the Certificate of Incorporation.

SECTION 2. *Special.* A special meeting may be called at any time by the President, or in case of his absence or disability by the President-Elect, and must be called at the request of a majority of the Council or fifty members of the Society. A notice specifying the purpose of such meeting shall be mailed to each member at least ten days previous thereto. The Council shall select the places at which all meetings shall be held.

SECTION 3. *Quorum.* Fifty members shall constitute a quorum at all meetings of the Society, but in the absence of a quorum any number shall be sufficient to adjourn to a fixed date.

ARTICLE III. *Officials*

SECTION 1. *Officers.* The officers shall be

a President, a President-Elect, a Secretary, and a Treasurer, who shall be elected annually by the members of the Society.

SECTION 2. *Council* A. The Officers so elected, and three additional members, one of whom shall be elected at each annual meeting of the Society to serve a three year term, shall constitute the Board of Directors of the corporation and shall be known as "The Council." The immediate Past-President shall be invited to meet with the Council but shall not have the power to vote.

B. No two members of the Council may be from the same institution, and none of the members shall be eligible for re-election until after the lapse of one year, except the Secretary and Treasurer who shall each be eligible for re-election for five successive years.

SECTION 3. *Duties of Officers.* The powers and duties of the officers elected by the Society shall be such as usually devolve upon their respective positions. The President-Elect shall act as President in case of the inability of the President to serve.

ARTICLE IV. *The Council*

SECTION 1. *Powers.* The general management of the Society during the intervals between meetings shall be vested in the Council, which shall regularly perform the ordinary duties of an executive committee and shall possess all of the powers conferred upon the Board of Directors of a membership corporation by the Membership Corporation Law of the State of New York.

SECTION 2. *Reports.* The Council shall report to the Society as promptly as possible its findings on the eligibility of candidates for membership and on all charges of a violation of these By-Laws.

ARTICLE V. *Nominations and Elections*

SECTION 1. *Nominating Committee.* A. The Nominating Committee shall consist of nine members from nine different institutions. Members who have served for two consecutive years shall be ineligible for re-election until after the lapse of one year. The members shall be elected at each annual meeting from a slate provided by the incumbent Nominating Committee, this slate to contain the names of at least fifteen Society members, each from a different institution, provided that this slate contain not more than five names of members of the incumbent Nominating Committee and further provided that to this slate there shall be added the name of any Society member received by the Secretary and supported by the petition of twenty or more Society members.

B. The nine nominees from different institutions who receive the highest number of votes on the official ballot shall be declared elected to the Nominating Committee, the one receiving the most votes being declared its Chairman and Secretary. Tie votes for the position of Chairman, or for the person in the ninth position in the report of the Tellers, shall be resolved by a vote cast by the President. The name of the person in tenth position shall be held as an alternate to serve in the event of the disability of one of the elected members other than the Chairman. In the event of the disability of the Chairman, the newly elected Nominating Committee shall elect its Chairman from among its own members including the alternate.

SECTION 2. *Nominations.* A. The Nominating Committee shall make not less than *two* nominations for the offices of the President-Elect and *two* for the position of Councilor, and at least one nomination for each of the remaining offices. In addition the Nominating Committee shall provide a slate of at least fifteen names of members of the Society selected as specified in Article V, Section 1 A, who shall be candidates for positions on the Nominating Committee of the following year. Furthermore, the Nominating Committee in each alternate year shall make not less than three nominations for positions on the Editorial Committee in accordance with the provisions of Article VII, Section 1 of these By-Laws. The names of nominees for each office shall be listed alphabetically on the ballot.

B. The nominations by the Nominating Committee must be transmitted to the Secretary at least two months before the annual meeting at which they are to be considered.

C. The Secretary shall prepare and send to every member, at least three weeks before the annual meeting, a printed ballot which shows the nominations of the Nominating Committee for the several offices and positions together with such other properly supported independent nominations for the Nominating Committee as may have been submitted by petition and received by him.

D. Nominations for membership on the Nominating Committee may be made by any member of the Society in the form of a petition addressed to the Secretary and to be received by him at least two months before the annual meeting. Such petitions must be supported by the signatures of not less than twenty members of the Society either attached to the petition or to accompanying documents.

SECTION 3. *Elections.* A. The members of the Society shall individually vote on the printed ballots presented to them by the Secretary and shall return them in signed envelopes to the Secretary not later than the hour set for the opening of the first executive session of the Annual Meeting.

B. At the time during the annual meeting that the election shall have been declared closed by the President, the Secretary shall present to the Tellers, appointed by the President to take charge of the election, all signed ballots received by him.

C. All elective positions shall be filled by ballot according to a report presented by the Tellers at an executive session of the annual meeting.

D. A *plurality* of the votes cast shall be necessary to elect, and in case of a tie vote the President shall cast the deciding ballot.

E. Elected officers shall take office on July 1st following the annual meeting.

SECTION 4. *Filling of Vacancies.* A. The Nominating Committee shall fill all vacancies in elective positions except such as may occur at a meeting of the Society. Any vacancy which occurs at a meeting shall be filled temporarily by vote of the Council.

B. The President of the Society shall fill all vacancies in appointive positions.

ARTICLE VI. *Financial*

SECTION 1. *Dues.* Annual assessments shall be determined by majority vote at the annual meetings, upon the recommendation of the Council. Members who have reached the age of 65 years, or who have become incapacitated, may request the consideration of the Council for exemption from payment of Society dues.

SECTION 2. *Expenditures.* No expenditures from the general funds of the Society except those required in the performance of the ordinary official duties shall be made except by vote of the Society or the Council or of its Finance Committee. Expenditures from any special funds which have accrued from taxation of the membership or otherwise shall require either the vote of the Council or of a Committee designated by the Council.

SECTION 3. *Privileges of Membership Begin with Payment of Dues.* Newly elected members are entitled to the privileges of membership only after payment of the dues following their election. Such dues shall be paid on or before July 1 of each year.

SECTION 4. *Penalty for Non-Payment of Dues.* A member who has not paid dues within a period of one year after billing shall, upon being billed for the succeeding year, be notified of forfeiture of membership unless all indebtedness to the Society is met within 60 days. Reinstatement shall require the request of the delinquent member, approval by the Council, and liquidation of all indebtedness.

SECTION 5. *Herter Fund.* A. *Use.* The "Christian A. Herter Memorial Fund" shall be held and invested separately from the general funds of the Society, in accordance with the terms of the agreement dated May 16, 1911, executed by the Journal of Biological Chemistry and the donors of said fund. The income thereof shall be expended under the direction of the Editorial Board exclusively for the maintenance and support of the Journal of Biological Chemistry subject to the supervision and control of the Finance Committee of the Council in accordance with the Trust Agreement.

B. *Trust Agreement.* The Council may from time to time make Agreement with a Trust Company which shall then serve as the depositary of the investments and income of the "Christian A. Herter Memorial Fund" hereinafter called the Herter Fund and of such other funds as the Society may from time to time commit to its charge. The Trust Company shall have and exercise the following powers and duties, viz., the custody and safekeeping of securities and cash belonging to the Herter Fund and other funds and the collection of income and other moneys due, with power to receipt for the same and to endorse for deposit all checks payable to the Society or the Treasurer, or to the Journal of Biological Chemistry for income or other moneys due to the Herter Fund or other funds; the investment or reinvestment of the capital of the Herter Fund or other funds, subject to the approval of the Council or the Chairman of the Finance Committee; the disbursement of principal under the direction of the Council and the disbursement of income of the Herter Fund under the direction of the Editorial Board of the Journal of Biological Chemistry with the approval of the Chairman of the Finance Committee of the Council. The Trust Company shall keep books of account and render statements, annually or oftener upon the request of the Council or its Finance Committee, setting forth the condition of the Herter Fund or other funds and the receipts and disbursements since the date of the preceding statement.

SECTION 6. *Finance Committee.* A. *Membership.* The Finance Committee shall consist of the President of the Society, the Treasurer of the Society, the Managing Editor of the Journal of Biological Chemistry, and the Chairman of the Editorial Committee *ex officiis* together with one member of the Society to be appointed by the Council for a period of five years. The President shall designate the Chairman annually from among its members.

B. *Duties.* The Finance Committee shall meet at the time of the annual meeting of the Society or at the call of the Chairman or President and shall examine and verify the records of the Trust Company and all financial records concerning the manufacture and sale of the Journal of Biological Chemistry, these records having been duly audited by an independent public accountant. They shall at all times act as the agents of the Council with respect to the financial acts of the Trust Company as defined in Sub-section B of Section 5 and the financial acts of the Managing Editor of the Journal of Biological Chemistry. They shall render an annual report to the Council.

ARTICLE VII. *Journal of Biological Chemistry*

SECTION 1. *Editorial Committee.* The Editorial Committee shall consist of nine members of the Society from different institutions who shall be nominated by the Nominating Committee

and elected by the Society in the same manner as the officers. The term of office shall be six years and members shall not be eligible for re-election until after an interval of one year. Three members shall be elected each alternate year to succeed the three members whose terms shall have expired.

SECTION 2. *Powers of Committee.* The Editorial Committee shall elect a Chairman from among its own membership and the Chairman shall be eligible for re-election. The Committee shall have the power to appoint the members of the Editorial Board from among the membership of the Society and to determine their term of office. In making these appointments, the recommendations of the Managing Editor of the Journal are to be taken into consideration with respect to the qualifications of the appointees and to the needs of the Journal. The Committee shall have the final authority in matters pertaining to the general policies of the Journal.

SECTION 3. *Editorial Board.* The members of the Editorial Board shall be appointed by the Editorial Committee to assist the Managing Editor in the editorial conduct of the Journal. The expenditure of the income of the Herter Fund shall be under the direction of the Editorial Board, subject to the provisions of Article VI, Section 5, of these By-Laws.

SECTION 4. *Managing Editor.* The Managing Editor shall be appointed by the Council in consultation with the Editorial Board and the Editorial Committee. He shall have direct responsibility and authority for both the active editorial conduct of the Journal and the management of its affairs, including contracts for publication, approval of advertising, office administration and related activities. The Managing Editor shall report annually to the Chairman of the Editorial Committee in advance of the regular meeting, and through him to the Council, on the condition of the Journal.

ARTICLE VIII. *Papers on Scientific Subjects*

SECTION 1. *Presentation of Papers.* The Council shall set specifications on the time allotment for presentation of papers by members at meetings of the Society. Members shall have the privilege of introducing non-members for the purpose of presenting papers unless the privilege is revoked or limited by the Council.

SECTION 2. *Number of Papers.* The Council shall, from time to time, set the rules on the number of papers which may be presented by a member, as necessitated by changing circumstances but with regard to equality of privilege for the members.

ARTICLE IX. *Corporate Seal*

SECTION 1. The corporate seal of the corporation shall be a circle surrounded by the words, "AMERICAN SOCIETY OF BIOLOGICAL CHEMISTS," and including the word, "INCORPORATED."

ARTICLE X. *Amendments*

SECTION 1. *Manner of Presentation.* Proposed amendments to the By-Laws must be sent to the Secretary at least three months before the date of the meeting at which they are to be considered and must be endorsed in writing by at least three members.

SECTION 2. *Notice of Intended Amendments.* The Secretary shall notify each member of proposed amendments at least three weeks before the meeting at which they are to be considered.

SECTION 3. *Adoption of Amendments.* Amendments to the By-Laws shall be voted upon by written ballot. After discussion at the first executive session, the Council shall instruct the Secretary whether to conduct the balloting at an executive session of the Society or by mail. Affirmative votes from three-fifths of the members voting shall be required for the adoption of an amendment. Amendments shall become effective July 1 following their adoption.

AMERICAN SOCIETY FOR PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS

Founded December 28, 1908; Incorporated June 19, 1933

OFFICERS, 1958–1959

President—C. D. Leake, Ohio State University, Columbus.

President-Elect—L. S. Goodman, University of Utah, Salt Lake City.

Past-President—Otto Krayer, Harvard Medical School, Boston, Massachusetts.

Treasurer—A. D. Bass, Vanderbilt University, Nashville, Tennessee.

Secretary—H. C. Hodge, University of Rochester, Rochester, New York.

Secretary-Elect—K. H. Beyer, Merck Sharp & Dohme Research Laboratories, West Point, Pennsylvania.

Council—C. D. Leake, L. S. Goodman, Otto Krayer, A. D. Bass, H. C. Hodge, K. H. Beyer, B. B. Brodie (1959), O. H. Lowry (1960), R. M. Featherstone (1961).

Historian—E. M. K. Geiling.

COMMITTEES

Membership Committee—W. F. Riker (1959), *Chairman;* J. A. Bain (1960), G. B. Koelle (1961).

Nominating Committee—G. H. Acheson, *Chairman;* B. E. Abreu, K. K. Chen, A. E. Farah, L. A. Woods.

Standing Committee on Educational Affairs—Lawrence Peters (1959), E. M. Boyd (1959), A. C. Bratton, Jr. (1959), A. D. Bass (1960), Klaus Unna (1960), K. H. Beyer, Jr. (1960), G. H. Acheson (1961), D. S. Riggs (1961).

Committee on Non-Scientific Professional Affairs—C. C. Pfeiffer (1959), G. B. Koelle (1959), A. E. Farah (1960), Alfred Gilman (1960), F. S. Philips (1961), Ernest Bueding (1961).

Program Committee—R. F. Furchgott (1959), N. C. Moran (1960), P. B. Dews (1961).

REPRESENTATIVES TO OTHER ORGANIZATIONS

American Association for the Advancement of Science—A. D. Bass (1959), E. J. de Beer (1959).

National Research Council—Division of Biology and Agriculture, A. J. Lehman (1959); Division of Medical Sciences, H. B. Haag (1959).

International Union of Physiological Sciences—C. F. Schmidt, M. H. Seevers, K. K. Chen, J. A. Shannon.

EDITORIAL BOARD

Board of Publication Trustees—Harry Beckman (1959), M. H. Seevers (1960), L. S. Goodman (1961), Lawrence Peters, Otto Krayer.

Journal of Pharmacology and Experimental Therapeutics—Lawrence Peters, *Editor;* Julius Axelrod, H. L. Borison, M. de V. Cotten, P. B. Dews, W. W. Douglas, K. P. Du Bois, Sydney Ellis, R. F. Furchgott, Avram Goldstein, Harris Isbell, C. J. Kensler, E. K. Killam, Louis Lasagna, N. C. Moran, G. H. Mudge, F. W. Schueler, C. C. Smith, J. L. Strominger, E. W. Sutherland, Jr., E. L. Way, T. C. West, L. A. Woods. *Index Editor*—R. M. Isenberger.

Pharmacological Reviews—Otto Krayer, *Chairman;* G. H. Acheson, H. Blaschko, G. A. H. Buttle, Erik Jacobsen, G. B. Koelle, Mark Nickerson, F. S. Philips, Börje Uvnäs, Marthe Vogt. *Editorial Advisory Board*—Adrien Albert, Eduardo Braun-Menéndez, F. von Brücke, K. Bucher, A. S. V. Burgen, J. A. Cohen, B. B. Dikshit, Pietro Di Mattei, Fernando Peres Gomez, P. Holtz, J. Jacob, Y. Kobayashi.

PAST OFFICERS

Presidents—**1909–1912** J. J. Abel. **1913–1915** T. Sollmann. **1916–1918** Reid Hunt. **1919–1920** A. S. Loevenhart. **1921–1923** C. W. Edmunds. **1924–1926** John Auer. **1927–1929** Carl Voegtlin. **1930–1931** G. B. Wallace. **1932–1933** W. de B. MacNider. **1934** R. A. Hatcher. **1935–1936** V. E. Henderson. **1937–1938** A. L. Tatum. **1939** O. H. Plant. **1940–1941** E. M. K. Geiling. **1942–1943** E. K. Marshall, Jr. **1944–1945** E. E. Nelson. **1946–1947** M. H. Seevers. **1948** C. A. Dragstedt. **1949–1950** C. F. Schmidt. **1951** McKeen Cattell. **1952** K. K. Chen. **1953** H. B. Haag. **1954** C. M. Gruber. **1955** E. G. Gross. **1956** Harry Beckman. **1957** Otto Krayer.

Vice-Presidents—**1932** A. N. Richards. **1933–1934** A. L. Tatum. **1935–1936** O. H. Plant. **1937–1939** E. M. K. Geiling. **1940–1941** C. F. Schmidt. **1942–1943** C. A. Dragstedt. **1944–1945** C. M. Gruber. **1946** H. B. Van Dyke. **1947** C. A. Dragstedt. **1948** H. B. Van Dyke. **1949** J. C. Krantz. **1950** McKeen Cattell.

Presidents-Elect—**1951** K. K. Chen. **1952** H. B. Haag. **1953** C. M. Gruber. **1954** E. G. Gross. **1955** Harry Beckman. **1956** Otto Krayer. **1957** C. D. Leake.

Secretaries—**1909–1911** Reid Hunt. **1912–1916** J. Auer. **1917** L. G. Rowntree. **1918–1930** E. D. Brown. **1931–1933** V. E. Henderson. **1934–1936** E. M. K. Geiling. **1937–1940** G. P. Grabfield. **1941–1945** R. N. Bieter. **1946–1950** H. B. Haag. **1951–1955** C. C. Pfeiffer. **1956–** H. C. Hodge.

Treasurers—**1909–1913** A. S. Loevenhart. **1914–1920** W. de B. MacNider. **1921–1923** H. McGuigan. **1924–1928** A. L. Tatum. **1929–1934** O. H. Plant. **1935–1938** C. M. Gruber. **1939–1943** E. E. Nelson. **1944–1946** McKeen Cattell. **1947–1950** K. K. Chen. **1951–1953** R. P. Walton. **1954–1957** J. M. Dille.

CONSTITUTION

ARTICLE I.

The name of this organization is the American Society for Pharmacology and Experimental Therapeutics, Incorporated.

ARTICLE II.

The purpose of this Society is to promote pharmacological knowledge and its use among scientists and to issue publications for this purpose.

ARTICLE III. *Membership*

SECTION 1. The Society shall consist of members and honorary members.

SECTION 2. A. Any qualified investigator who has conducted and published a meritorious original investigation in pharmacology shall be eligible for membership in the Society, subject to the conditions set forth in the by-laws.

SECTION 2. B. Distinguished scientists of any country who have contributed to the advance of pharmacological science shall be eligible for proposal as honorary members of the Society, subject to the conditions set forth in the by-laws.

SECTION 3. The Council, for appropriate reasons, may decide that it is for the best interests of the Society that a member or an honorary member be expelled. In this case, the member shall be notified and given an opportunity for a hearing before the Council. If a majority of the members of the Council favor expulsion, the Council shall so recommend to the Society. A vote of three-fourths of the members present at an executive session of the Society shall be required for expulsion. Subsequent reinstatement shall follow the course prescribed for candidates for membership.

ARTICLE IV. *Officers*

SECTION 1. The management of the Society shall be vested in a Council consisting of the President, the President-Elect, the immediate Past-President, the Secretary, the Treasurer, the Secretary-Elect or the Treasurer-Elect, and three other Councilors.

SECTION 2. The President-Elect, either the Secretary-Elect or the Treasurer-Elect, and one other Councilor shall be elected at each annual meeting and shall assume office on July 1 or January 1, whichever occurs first after their election.

SECTION 3. The President-Elect shall become President after one year as President-Elect or upon the earlier death or resignation of the President, and two years after assuming office as President-Elect, he shall become Past-President for one year.

SECTION 4. The Councilors shall serve for three years.

SECTION 5. A Secretary-Elect and a Treasurer-Elect shall be elected in alternate years to serve for one year before taking over the post as Secretary and Treasurer, respectively, for a term of two years.

SECTION 6. Vacancies on the Council or committees which occur between annual meetings shall be filled by a majority vote of the Council. The persons so chosen shall serve until the next meeting of the Society, at which time the vacancies shall be filled by election.

ARTICLE V. *Standing Committees*

SECTION 1. There shall be a Membership Committee consisting of three members of the Society who hold no other elective office in the Society. One shall be elected at each annual meeting and serve for three years.

SECTION 2. There shall be a Nominating Committee consisting of five members of the Society who hold no other office in the Society. All shall be nominated and elected at the annual meeting. They shall serve for one year.

SECTION 3. A. The President, after consultation with the Council and subject to approval by the Society at its next executive session, shall appoint one member of the Board of Publications Trustees each year, and shall desinate the Chairman of the Board.

SECTION 3. B. The Board shall elect an Editor for each official journal of the Society. Each Editor shall act as an additional voting member of the Board in all its activities except election of Editors of the journals.

SECTION 3. C. The Board shall be vested with full power from the Society to control and manage, both editorially and financially, all publications of the Society. The Chairman of the Board shall be an *ex officio* member of the Council without vote and shall keep the Council informed of the major activities of the Board. He shall report annually to the Society at an executive session.

SECTION 3. D. The Editor of each journal may nominate Associate Editors for election by the Board.

SECTION 3. E. The members of the Board

and the Associate Editors shall serve for three years, are eligible for reappointment, and may hold office concurrently in the Society.

ARTICLE VI.

Except as provided in the by-laws, the annual meeting or other meetings of the Society shall be held at times and places determined by the Council. At least four weeks before such meetings, notice of their time and place shall be sent to each member of the Society.

ARTICLE VII.

SECTION 1. Beyond the expenditures required by the ordinary activities of the Society, no money from the general Society account shall be disbursed save by authority of the Council or the Society.

SECTION 2. The annual assessment shall be determined by majority vote at an executive session of the Society, upon recommendation of the Council. It shall become due on the following July 1 unless another time is recommended by the Council and approved by the Society.

SECTION 3. The Treasurer shall give a financial report at the annual meeting.

SECTION 4. All publication funds shall be kept in a separate account, subject to the control of the Board of Publications Trustees. No publication funds may be diverted for other than publication purposes without the approval of the Society upon joint recommendation of the Board and the Council. The Board shall make an annual financial report to the Society at an executive session.

ARTICLE VIII.

Fifty members shall constitute a quorum for the transaction of business.

ARTICLE IX.

SECTION 1. Intended amendments to the Constitution, or certificate of incorporation, may be acted upon at any executive session of the Society, provided that each proposal is endorsed in writing by at least three members and is placed in the hands of the President not less than two months before the meeting. At least four weeks before the meeting at which amendments are to be considered, the proposed amendments and notice of the time and place at which they will be considered shall be sent to each member. A four-fifths vote of the members present shall be required for the adoption of an amendment to the Constitution or certificate of incorporation.

SECTION 2. Adoption, alteration, or repeal of by-laws may be moved at any executive session of the Society. Their passage shall require a two-thirds vote of the members present in each of two executive sessions of the Society on separate days.

BY-LAWS

I. *Members*

1. Except as hereinafter provided, after 1958 only persons having legal residence in the United States, its dependencies, Canada, and Mexico shall be eligible for election as members of the Society.

2. In special cases, candidates for membership from areas other than those described in By-Law I.1. may be proposed by the Membership Committee or by the Council. These candidates must be unanimously approved by the Council and the Membership Committee; their election by the Society shall follow the rules prescribed for members.

3. Candidates for membership shall be proposed by two members of the Society, who are not members of the Council, at least ten months before the annual meeting, on forms provided by the Secretary.

4. The Membership Committee shall investigate the qualifications of the candidates for membership and report to the Council at least four months prior to the annual meeting.

5. The Council may recommend for election as members the candidates reported to them provided they have been approved by two-thirds of the combined members present of the Membership Committee and the Council.

6. The names of the candidates recommended for admission by the Council shall be posted not later than the day preceding the election of members.

7. New members shall be elected at the annual meeting. The election shall be by individual, secret ballot. One opposing vote in every eight cast shall be enough to exclude a candidate from membership.

8. Nomination of honorary members shall follow the rules prescribed for members. Election of honorary members shall require the unanimous vote of the members present.

9. Honorary members shall have the privileges of membership but are not subject to the annual assessment.

10. Any member of the Society who has been a member for thirty years, or who has retired because of disability or age, may upon approval of the Council be relieved from the annual assessment. He shall retain the privileges of membership.

11. Any member whose assessment is three years in arrears shall cease to be a member of the Society, unless he is reinstated by a vote of the Council. The delinquent shall be notified of his impending disqualification and informed of his right to appeal to the Council for reinstatement.

II. *Nomination and Election*

1. Only members shall be eligible for elec-

tion or appointment to the Council and Standing Committees of the Society.

2. No person who has served as President more than 3 months is eligible for re-election as President-Elect.

3. Persons elected as Councilors shall not be eligible for immediate re-election on expiration of their term of office.

4. The Executive Secretary shall not hold elective office in the Society.

5. No two members of the Membership Committee shall be from the same institution. None shall be eligible for immediate re-election.

6. No two members of the Nominating Committee shall be from the same institution. Members of the Nominating Committee are eligible for re-election for only one additional consecutive term.

7. The Nominating Committee shall make at least two nominations for each elective office except the Nominating Committee. At least four weeks before the annual meeting, these nominations shall be sent to each member together with notification of the time and place of the election.

8. Additional nominations may be made from the floor at the annual meeting, during the session at which the election occurs.

9. At least ten nominations for members of the Nominating Committee shall be made from the floor during the session at which the election occurs. The five nominees who receive the highest number of votes shall be declared elected.

10. At the first executive session of the annual meeting, each member present shall be given a printed ballot showing the nominations by the Nominating Committee and providing spaces for adding nominations made from the floor. A preliminary vote will be made from these ballots, and the tellers, appointed by the President, shall as soon as possible post a list showing the two nominees for each office who have received the highest number of votes. A final vote between these pairs of candidates shall be taken at the close of the first executive session of the annual meeting. A majority of the votes cast shall be necessary for election. Both of the said votes shall be by secret ballot.

III. *Officers and Committees*

1. The Council may appoint and compensate an Executive Secretary to assist it in carrying on the functions of the Society, including receipt and disbursement of funds under direction of the Council.

2. That member of the Membership Committee who has served the longest current consecutive period shall be the chairman of the Committee.

3. The member of the Nominating Committee elected by the highest number of votes shall be the chairman.

4. The Board of Publications Trustees shall meet at least once a year. Three members shall constitute a quorum for the conduct of business.

5. Only members of the Society are eligible as Editors or Associate Editors of the Society's journals, except that foreign non-members may be Associate Editors of international journals of the Society.

6. Non-members of the Society may be appointed to editorial advisory committees in connection with the activities of the Society's journals.

IV. *Meetings*

1. The annual meeting of the Society shall be the meeting held in conjunction with the Federation of American Societies for Experimental Biology.

2. The Council shall designate one or more of its members to arrange the scientific programs and may appoint a Program Committee to assist in this task.

3. The Council shall establish regulations governing the presentation of papers at scientific sessions. These regulations shall be sent to each member at least three months before any meeting at which they are to apply.

4. Executive sessions of the Society may be held at any of its meetings.

5. The Council shall insure that only members attend and cast votes in the executive sessions of the Society.

6. The rules contained in O. Garfield Jones, Parliamentary Procedure at a Glance, Appleton-Century-Crafts, Inc., New York, 1949 edition, shall govern the conduct of the executive sessions of the Society when they are applicable and are not inconsistent with this Constitution and By-Laws or with special rules of order adopted by the Society.

THE AMERICAN SOCIETY FOR EXPERIMENTAL PATHOLOGY

Founded December 29, 1913; Incorporated August 31, 1950

OFFICERS, 1958–1959

President–William B. Wartman, Northwestern University, Chicago 11, Illinois.

Vice-President–C. C. Erickson, University of Tennessee, Memphis 3.

Past-President–Emory D. Warner, State University of Iowa, Iowa City.

Secretary-Treasurer–J. F. A. McManus, University of Alabama Medical Center, Birmingham.

Council–W. B. Wartman, C. C. Erickson, E. D. Warner, J. F. A. McManus, Jacob Furth (1957–59), K. M. Brinkhous (1958–60).

SPECIAL COMMITTEES

Meritorious Award or Honors Committee–D. E. Sprunt, *Chairman;* W. H. Carnes, F. W. Hartman, T. D. Kinney, A. J. Cox, Jr.

Publications Committee–R. B. Jennings, *Chairman;* J. F. A. McManus, J. J. Lalich.

Membership Survey Committee–C. E. Dunlap, *Chairman;* W. T. Hill, Simon Koletsky, S. C. Sommers, C. L. Yuile.

Nutritional Pathology Committee–R. L. Holman, *Chairman;* R. H. Follis, Jr., C. L. Pirani, W. S. Hartroft, A. J. Cox, Jr., H. L. Radcliffe.

REPRESENTATIVES TO OTHER ORGANIZATIONS

National Research Council–Division of Biology and Agriculture, Hans Schlumberger; Division of Medical Sciences, R. L. Holman.

American Association for the Advancement of Science–E. D. Warner.

Eli Lilly Award Committee (Joint with the Society of American Bacteriologists)–*Nominations:* W. H. Carnes; *Awards:* F. J. Dixon.

Commission for Biological Stains–J. F. A. McManus.

Intersociety Committee on Increasing Research Potential–C. C. Erickson.

Intersociety Committee on Public Information and Relations–F. W. Hartman.

PAST OFFICERS

Presidents–**1914** R. M. Pearce. **1915** Theobald Smith. **1916** Simon Flexner. **1917** Ludvig Hektoen. **1918** H. G. Wells. **1919** W. G. MacCallum. **1920** W. H. Park. **1921** F. G. Novy. **1922** H. T. Karsner. **1923** E. L. Opie. **1924** A. S. Warthin. **1925** G. H. Whipple. **1926** W. H. Brown. **1927** David Marine. **1928** E. B. Krumbhaar. **1929** W. F. Petersen. **1930** F. L. Gates. **1931** S. R. Haythorn. **1932** Peyton Rous. **1933** C. V. Weller. **1934** S. B. Wolbach. **1935** Oskar Klotz. **1936** A. R. Dochez. **1937** C. P. Miller. **1938** Morton McCutcheon. **1939** E. W. Goodpasture. **1940** Shields Warren. **1941** J. L. Bollman. **1942–1945** Balduin Lucké. **1946** P. R. Cannon. **1947** D. H. Sprunt. **1948** H. P. Smith. **1949** J. G. Kidd. **1950** J. F. Rinehart. **1951** F. S. Robscheit-Robbins. **1952** S. C. Madden. **1953** D. Murray Angevine. **1954** R. L. Holman. **1955** H. L. Stewart. **1956** F. W. Hartman. **1957** E. D. Warner.

Vice-Presidents–**1914** J. S. Anderson. **1915** G. H. Whipple. **1916–1917** Leo Loeb. **1918** W. G. Mac Callum. **1919** W. H. Park. **1920** F. G. Novy. **1921** H. T. Karsner. **1922** E. L. Opie. **1923** A. S. Warthin. **1924** G. H. Whipple. **1925** W. H. Brown. **1926** David Marine. **1927** E. B. Krumbhaar. **1928** W. F. Petersen. **1929** F. L. Gates. **1930** S. R. Haythorn. **1931** Peyton Rous. **1932** C. V. Weller. **1933** S. B. Wolbach. **1934** Oskar Klotz. **1935** A. R. Dochez. **1936** C. P. Miller. **1937** Morton McCutcheon. **1938** E. W. Goodpasture. **1939** Shields Warren. **1940** J. L. Bollman. **1941** Balduin Lucké. **1942–1945** P. R. Cannon. **1946** D. H. Sprunt. **1947** H. P. Smith. **1948** J. G. Kidd. **1949** J. F. Rinehart. **1950** F. S. Robscheit-Robbins. **1951** S. C. Madden. **1952** D. M. Angevine. **1953** R. L. Holman. **1954** H. L. Stewart. **1955** F. W. Hartman. **1956** E. D. Warner. **1957** W. B. Wartman.

Secretary-Treasurers–**1914** G. H. Whipple. **1915–1916** Peyton Rous. **1917–1920** H. T. Karsner. **1921–1923** W. H. Brown. **1924–1926** E. B. Krumbhaar. **1927–1929** C. V. Weller. **1930–1933** C. P. Miller. **1934–1936** Shields Warren. **1937–1939** P. R. Cannon. **1940–1945** H. P. Smith. **1946–1949** F. S. Robscheit-Robbins. **1949–1950** S. C. Madden. **1951–1952** R. L. Holman. **1953–1957** C. C. Erickson. **1957** J. F. A. McManus.

CONSTITUTION

ARTICLE I. *Name*

The Society shall be named "THE AMERICAN SOCIETY FOR EXPERIMENTAL PATHOLOGY."

ARTICLE II. *Object*

The object of this Society is to bring the productive investigators in pathology, working essentially by experimental methods, in closer

affiliation with the workers in the other fields of experimental medicine.

ARTICLE III. *Time and Place of Meeting*

The Society shall meet at the same time and place as the Federation of American Societies for Experimental Biology, which comprises at present the American Physiological Society, the American Society of Biological Chemists, the American Society for Pharmacology and Experimental Therapeutics, the American Society for Experimental Pathology, the American Institute of Nutrition and the American Association of Immunologists.

ARTICLE IV. *Membership*

SECTION 1. Any American investigator who, through the use of experimental methods, has, within three years prior to his candidacy, contributed meritorious work in pathology, is eligible to membership.

SECTION 2. It shall be the policy of the Society to restrict its membership to as small numbers as is compatible with the maintenance of an active existence.

SECTION 3. There shall be two classes of members: active and honorary members.

Active members: Candidates for active membership shall be nominated at or before an annual meeting by two members of the Society. The nominators shall present to the Secretary in writing evidence of the candidate's qualifications for membership. Nominations approved by the Council shall be presented to the Society for election at the next annual meeting following nomination. For election a favorable ballot by a majority of the members present is necessary.

Honorary members: These may be elected from the active list or from the group of distinguished investigators at home or abroad who have contributed to the knowledge of pathology by experimental study. They shall be elected only by the unanimous vote of the members present at time of nomination.

SECTION 4. Active members shall pay such annual dues as are determined upon, from year to year, by the Council. Honorary members shall pay no dues, are not eligible to office, and have no vote in the business affairs of the Society, but they shall have all the privileges of the active members in the scientific proceedings.

SECTION 5. Upon failure of an active member to pay dues for two years, notice shall be given to the member by the Secretary. At the end of the third year, if dues are still unpaid, such failure constitutes forfeiture of membership.

SECTION 6. A motion for expulsion of a member must be thoroughly investigated by the Council; at this investigation the accused shall be afforded a hearing or may be represented by a member. Expulsion can be accomplished only after a unanimous vote by the Council in favor of expulsion, sustained by a four-fifths vote of the members present at the meeting.

ARTICLE V. *Officers*

The management of the Society shall be vested in a Council of six members, consisting of a President, the Past President, a Vice-President, a Secretary-Treasurer, and two other members who shall be nominated by the Council and elected by the Society. Officers are elected by a majority vote and remain in office until July 1 following the Federation Meeting. Vacancies shall be filled by the Council for the unexpired term.

The President and Vice-President shall hold office for one year and are ineligible for re-election during the following year. The Secretary-Treasurer is eligible for re-election. Councilors shall hold office for two years and are elected on alternate years. At the first election one Councilor shall be elected for a short term of one year.

ARTICLE VI. *Quorum*

SECTION 1. Three constitute a quorum of the Council. The Council decides by a majority vote.

SECTION 2. A quorum of the Society for transaction of business shall be 30 active members. In all questions brought before the Society a majority vote of those present shall decide, except as elsewhere provided for.

ARTICLE VII. *Annual Meeting*

SECTION 1. Papers shall be limited to ten minutes. However, on motion and with unanimous consent, the time may be prolonged by a period not exceeding five minutes. The Council may make provision for longer papers on suitable occasions.

SECTION 2. The subjects of papers must be confined to experimental work in pathology. In doubtful cases a liberal interpretation by the President and Secretary may prevail. The Council may invite, however, presentations dealing with any subject which it considers of considerable interest to the Society.

ARTICLE VIII. *Change of Constitution*

A motion concerning a change of the Constitution must be presented to the Council in writing by three members, and must be communicated to the members by the Secretary at least four weeks before the annual meeting. At this meeting such a change may be established when accepted by a four-fifths vote of the members present.

BY-LAWS

1. There must be in each year at least one meeting of the Council, which shall take place not later than the evening before the annual meeting.

2. At the end of the first session of the annual meeting the Secretary shall read the report of the Council. This report shall include (1) names of persons recommended for membership, (2) nominations for offices, (3) matters of general interest. The Secretary shall exhibit in a conspicuous place the names of candidates for membership recommended by the Council, together with the evidence of the qualifications of the candidates.

3. The election of officers and of new members, changes in the Constitution, etc., shall be voted upon at the end of the first session.

4. Changes in the By-Laws may be determined by a majority vote of those present.

5. In the year that a new Secretary-Treasurer is elected the incoming Council Member elected that year, or another member of the Council, shall become Assistant Secretary-Treasurer for the duration of the term of the Secretary-Treasurer.

AMERICAN INSTITUTE OF NUTRITION

Founded April 11, 1933; Incorporated November 16, 1934; Member of Federation 1940

OFFICERS, 1958–1959

President—W. J. Darby, Vanderbilt University, Nashville, Tennessee.

President-Elect—D. W. Woolley, Rockefeller Institute for Medical Research, New York City.

Past-President—R. R. Williams, 297 Summit Ave., Summit, New Jersey.

Secretary—G. M. Briggs, National Institutes of Health, Bethesda 14, Maryland (1960).

Treasurer—J. B. Brown, Ohio State University, Columbus (1959).

Council—W. J. Darby, D. W. Woolley, R. R. Williams, J. B. Brown, G. M. Briggs, E. W. McHenry (1959), Paul György (1960), J. H. Roe (1961).

STANDING COMMITTEES

Nominating Committee—H. H. Williams, *Chairman*; Gladys Emerson, R. W. Engel, J. M. Orten, O. L. Kline.

Committee on Membership Qualifications—A. E. Harper, *Chairman*; R. M. Leverton, C. M. Lyman, A. E. Schaefer, F. J. Stare.

Committee on Nomenclature (Joint with American Society of Biological Chemists)—O. L. Kline, *Chairman*; P. L. White.

Committee on Journal of Nutrition—D. M. Hegsted, *Chairman*; R. W. Engel, O. L. Kline, M. O. Lee, C. V. Moore, B. L. Oser, E. E. Snell, C. M. Young.

Borden Award Nominating Committee—H. H. Williams (1959), *Chairman*; C. A. Baumann (1960), C. V. Mann (1961).

Osborne-Mendel Award Nominating Committee—I. M. Hoobler (1959), *Chairman*; D. M. Hegsted (1960), J. S. Dinning (1961).

Fellows Committee—T. H. Jukes (1959), *Chairman*; Paul György (1960), C. G. Mackenzie (1960), W. D. Salmon (1961), Karl Folkers (1961).

Public Information Committee—R. W. Engel (1960), *Chairman*; G. M. Briggs (1960), LeRoy Voris (1960), A. E. Schaefer (1960).

Auditing Committee—F. A. Hitchcock (1959), *Chairman*; M. B. Patton (1959).

Tellers Committee—C. D. Tolle, *Chairman*; E. O. Keiles.

U. S. National Committee–IUNS (Appointed Members)—Paul György (1961), *Chairman*; W. H. Sebrell, Jr. (1961), *Vice-Chairman*; E. N. Todhunter (1959), A. E. Schaefer (1959), G. F. Combs (1959), LeRoy Voris (1960), M. O. Lee (1960), R. W. Engel (1960), E. L. Severinghaus (1961).

Fifth International Congress on Nutrition, 1960, Officers and Committees (Organized by American Institute of Nutrition and U. S. National Committee of IUNS)—E. V. McCollum, *Honorary President*; C. G. King, *President. Organizing Committee*: Paul György, *Chairman*; M. O. Lee, *General Secretary*; G. M. Briggs, *Recording Secretary*; W. H. Sebrell, Jr., *Chairman of Program Committee*; E. L. Severinghaus, *Chairman of Finance and Budget Committee*; F. S. Daft, *Chairman of Hospitality Committee (and Co-Chairman of Finance and Budget Committee)*; W. J. Darby, *Chairman of Lectureships and Travel Assistance Committee*; Hazel Stiebeling, *Chairman of Publications Committee*; LeRoy Voris, *Chairman of Public Information Committee; Other Members:* C. H. Best, C. A. Elvehjem, Gladys Emerson, R. W. Engel, Grace Goldsmith, F. Gomez-S, L. A. Maynard, E. M. Nelson, L. B. Pett, H. E. Robinson, R. R. Williams.

REPRESENTATIVES TO OTHER ORGANIZATIONS

National Research Council Boards and Divisions—N. R. Ellis (1960).

American Association for the Advancement of Science—To Section C, H. A. Schneider (1960); to Section N, P. L. Day (1959).

Food and Agricultural Organization—J. M. Hundley (1960).

Editorial Board, Journal of Nutrition

George R. Cowgill (1959), *Editor;* J. S. Dinning (1959), J. M. Orten (1959), C. R. Treadwell (1959), C. P. Berg (1960), C. G. Mackenzie (1960), H. R. Bird, (1960), R. W. Engel (1961), P. L. Harris (1961), H. A. Schneider (1961), O. L. Kline (1962), E. S. Nasset (1962), Herbert Pollack (1962).

PAST OFFICERS

(Year Elected)

Presidents—**1933** L. B. Mendel. **1934–1935** J. R. Murlin. **1936** E. F. DuBois. **1937** Mary S. Rose. **1938** E. V. Mc Collum. **1939** H. C. Sherman. **1940** T. M. Carpenter. **1941** A. G. Hogan. **1942** L. A. Maynard. **1943** H. B. Lewis. **1944** Icie G. Macy-Hoobler. **1945** W. C. Rose. **1946** A. H. Smith. **1947** R. M. Bethke. **1948** E. M. Nelson. **1949** C. G. King. **1950** W. H. Griffith. **1951** C. M. McCay. **1952** Paul L. Day.

1953 C. A. Elvehjem. **1954** G. R. Cowgill. **1955** W. H. Sebrell, Jr. **1956** H. J. Deuel, Jr. **1957** R. R. Williams.

Vice-Presidents—**1933** H. C. Sherman. **1934-1935** E. F. DuBois. **1936** Mary S. Rose. **1937** E. V. McCollum. **1938–1939** T. M. Carpenter. **1940** A. G. Hogan. **1941** L. A. Maynard. **1942** H. B. Lewis. **1943** Icie G. Macy-Hoobler. **1944** W. C. Rose. **1945** A. H. Smith. **1946** R. M. Bethke. **1947** E. M. Nelson. **1948** C. G. King. **1949** W. H. Griffith. **1950** C. M. McCay. **1951** P. L. Day. **1952** C. A. Elvehjem. **1953** George R. Cowgill. **1954** W. H. Sebrell, Jr. **1955** H. J. Deuel, Jr. **1956** R. R. Williams. **1957** W. J. Darby.

Secretaries—**1933** J. R. Murlin. **1934** Icie G. Macy. **1938** L. A. Maynard. **1941** A. H. Smith. **1945** H. E. Carter. **1948** J. H. Roe. **1951** J. M. Orten. **1954** R. W. Engel. **1957** G. M. Briggs.

Treasurers—**1933** J. R. Murlin. **1934** W. M. Boothby. **1935** G. R. Cowgill. **1940** W. H. Sebrell, Jr. **1944** E. M. Nelson. **1947** N. R. Ellis. **1950** N. B. Guerrant. **1953** O. L. Kline. **1956** J. B. Brown.

CONSTITUTION

1. The name of the Society is the "AMERICAN INSTITUTE OF NUTRITION."

2. The purposes of the Society are to further the extension of the knowledge of nutrition and to facilitate personal contact between investigators in nutrition and closely related fields of interest.

3. The management of the American Institute of Nutrition shall be vested in a council consisting of the President, President-Elect, Past President, Secretary, Treasurer and three additional members.

BY-LAWS

ARTICLE I. *Membership*

SECTION 1. *Eligibility for membership:* Members. Qualified investigators who have independently conducted and published meritorious original investigations in some phase of the chemistry or physiology of nutrition and who have shown a professional interest in nutrition for at least 5 years shall be eligible for membership in the Society.

SECTION 2. *Nomination:* Nominations for membership shall be made and seconded by members of the Society on blanks furnished by the Secretary. Nominations shall be submitted to the Council who shall determine eligibility and make recommendation to the Society at a regular meeting.

SECTION 3. *Election to membership:* A. A nominee for membership may be voted for by ballot at any meeting of the Society after the Council has reported its findings on his eligibility. B. A majority of the ballots cast shall elect.

SECTION 4. *Forfeiture:* If a majority of the Council after due notice to the member in question and opportunity for a hearing, shall decide that the interests of the Society require the expulsion of a member, the Secretary shall send a notice of this decision to each member at least two weeks before the next annual meeting. At this meeting the Secretary shall, on behalf of the Council, propose the expulsion; and if two-thirds of the members present vote for it, the member shall be expelled, his assessment for the current year shall be returned to him, and he shall cease to be a member of the Society.

SECTION 5. *Fellows:* Active or retired members of the American Institute of Nutrition who have passed their sixty-fifth birthday and who have had a distinguished career in nutrition may be selected as Fellows of the Society. Such selection shall be made by a Fellows Committee of five members appointed by the incoming President. Nominations of candidates may be made to the Fellows Committee by any member of the Society. No more than three Fellows shall be selected each year (except that a maximum of ten may be selected the first year this provision is put into effect). A suitable citation shall accompany awarding of Fellowships at the annual meeting.

ARTICLE II. *Meetings and Quorum*

SECTION 1. *Annual:* The annual meeting of the Society shall be held on the date fixed by the Certificate of Incorporation.

SECTION 2. *Special:* A special meeting may be called at any time by the President, or in case of his absence or disability, by the President-Elect, and must be called at the request in writing of a majority of the Council or fifty members of the Society. Notice specifying the purpose of such meeting shall be mailed to each member at least ten days previous thereto. The Council shall select the places at which meetings shall be held.

SECTION 3. *Quorum:* Thirty members shall constitute a quorum at all meetings of the Society, but in the absence of a quorum any number shall be sufficient to adjourn to a fixed date.

ARTICLE III. *Officials*

SECTION 1. *Officers:* The officers, elected by members of the Society by majority vote, shall be a President, a President-Elect to be elected annually, and a Secretary and a Treasurer each of whom shall be elected to serve for a term of three years. Their terms of office shall commence on July 1 of the year in which they are elected. No person who has served as President is eligible for re-election as President-Elect.

SECTION 2. *Council:* The officers so elected, the Past President and three additional members, one of whom shall be elected at each annual

meeting to serve a term of three years, shall constitute a Board of Trustees and shall be known as "The Council."

SECTION 3. *Duties of officers:* The powers and duties of the officers elected by the Society shall be such as usually devolve upon their respective positions.

ARTICLE IV. *The Council*

SECTION 1. *Powers:* The general management of the Society during the intervals between meetings shall be vested in the Council, which shall regularly perform the ordinary duties of an executive committee and possess all the powers conferred upon the Board of Trustees of an educational institution chartered by the Education Department of the University of the State of New York. A permanent charter was issued to the American Institute of Nutrition under date of November 16, 1934.

SECTION 2. *Reports:* The Council shall report to the Society its findings on the eligibility of candidates for membership, and on all charges of a violation of these By-Laws.

ARTICLE V. *Officer Nominating Committee and Elections*

SECTION 1. *Membership:* A. The Nominating Committee shall consist of five members appointed for the coming year by the retiring President. Members who have served on the Nominating Committee for two consecutive years shall be ineligible for reappointment until after a lapse of one year. B. The President shall designate one member to be Chairman of the Nominating Committee.

SECTION 2. *Nomination of officials:* A. The Nominating Committee shall make at least one nomination for each of the offices to be filled, for each of the additional positions on the Council to be filled, and for each of the positions on the Editorial Board to be filled at the time of the annual meeting. Any member of the Institute may submit nominations to the Nominating Committee for its consideration along with those nominations made by the members of the Nominating Committee. B. The nominations by the Nominating Committee shall be transmitted to the Secretary at least six weeks before the annual meeting at which they are to be considered. C. The Secretary shall send to every member, at least two weeks before the annual meeting, a printed ballot containing the list of nominees and space for such additional names as the member wishes to propose, and at the same time shall notify the members that they may vote by mail, returning to the Secretary the marked ballot in the envelope provided, at such a time and place as the Secretary may designate, or the ballot may be delivered to the Secretary at the beginning of the business session at which the elections are to take place.

SECTION 3. *Election of officials:* A. At the beginning of the business session the Secretary shall present to the tellers, appointed by the President, the ballots submitted by the members and the ballots shall be counted forthwith. B. A majority of votes cast shall be necessary to elect an official.

SECTION 4. *Filling of vacancies:* A. Should a vacancy occur in any elective position, the Council, by majority vote, shall have the power to appoint a member of the Society to fill the unexpired term until the following election. B. The President of the Society shall fill all vacancies in appointive positions.

ARTICLE VI. *Financial*

SECTION 1. *Dues:* The dues shall be the annual cost of subscription to The Journal of Nutrition for members plus an annual assessment which shall be determined by majority vote at the annual meetings, upon recommendation of the Council, and shall be due within a month after the annual meeting. A member on attaining the age of 65 may elect to be relieved from all financial obligations to the Institute including subscription to The Journal of Nutrition.

SECTION 2. *Expenditures:* No expenditures from the general funds of the Society except those required in the performance of the ordinary official duties shall be made except by vote of the Society or the Council.

SECTION 3. *Penalty for non-payment of dues:* A. Members in arrears for dues for two consecutive years shall forfeit their membership. B. Delinquent members may be reinstated by the Council provided all indebtedness to the Society is liquidated.

SECTION 4. *Auditing Committee:* An Auditing Committee of two members, appointed for the coming year by the incoming President, should be responsible for auditing the records of the Treasurer and for submitting a written report at the next annual meeting. One member shall be designated as Chairman and members are eligible for reappointment up to a total of three years.

ARTICLE VII. *The Journal of Nutrition*

SECTION 1. The American Institute of Nutrition designates *The Journal of Nutrition* as its official organ of publication.

SECTION 2. In accordance with the expressed wish of the Wistar Institute of Anatomy and Biology, owner and publisher of *The Journal of Nutrition,* the American Institute of Nutrition shall nominate members of the Editorial Board for its official organ. A. The editorial management of *The Journal of Nutrition* shall be vested in an Editorial Board consisting of an Editor and twelve Board Members. B. The Editor shall be chosen by the Editorial Board to serve a term of five years beginning July 1 of the year in which he is chosen, and shall be eligible for re-election. The Editor shall have the power to

designate one of the Board Members to serve as his assistant, and such an appointee shall be called Associate Editor. C. Three members of the Institute shall be nominated by the Nominating Committee for membership on the Editorial Board each year to serve a term of four years, replacing three retiring members and taking office May 1 of the year in which they are elected. D. Retiring members of the Editorial Board shall not be eligible for renomination until one year after their retirement.

ARTICLE VIII. *Papers on Scientific Subjects*

SECTION 1. The Secretary shall be authorized to arrange programs for the scientific sessions at the annual meetings.

SECTION 2. The Council shall, from time to time, set the rules on the number of papers which may be presented or sponsored by a member, as necessitated by changing circumstances but with regard to equality of privilege for all members.

ARTICLE IX. *Changes in Constitution and By-Laws*

SECTION 1. Proposed changes in the Constitution and By-Laws must be sent in writing to the Secretary at least one month before the date of the meeting at which they are to be considered, and must be signed by at least three members. The Secretary shall send a printed copy of any proposed change to each member at least two weeks before the next meeting and shall notify all members that they may vote by proxy.

SECTION 2. If at this meeting two-thirds of the votes cast shall favor the proposed change, it shall be made.

THE AMERICAN ASSOCIATION OF IMMUNOLOGISTS

Founded June 19, 1913; Member of Federation 1942

OFFICERS, 1958–1959

President–J. E. Smadel, National Institutes of Health, Bethesda 14, Maryland.

Honorary President–A. F. Coca, Oradell, New Jersey.

Vice-President–W. C. Boyd, Boston University School of Medicine, Boston, Massachusetts.

Secretary-Treasurer–Calderon Howe, Columbia University College of Physicians & Surgeons, New York City.

Council–J. E. Smadel, A. F. Coca, W. C. Boyd, Calderon Howe, A. H. Coons, Rebecca Lancefield, Werner Henle, F. S. Cheever, J. Y. Sugg.

REPRESENTATIVES TO OTHER ORGANIZATIONS

National Research Council–Division of Medical Sciences, C. P. Miller.

American Association for the Advancement of Science–Lawrence Kilham, D. T. Smith.

Eli Lilly Award Committee (Joint with Society of American Bacteriologists)–*Nominations:* Sidney Raffel; *Awards:* S. S. Elberg.

Editorial Board, Journal of Immunology

J. Y. Sugg (1959), *Editor-in-Chief;* Jules Freund (1959), Geoffrey Edsall (1959), W. C. Boyd (1961), C. M. MacLeod (1961), Werner Henle (1962), F. L. Horsfall, Jr. (1962).

PAST OFFICERS

Presidents–**1913** G. B. Webb. **1915** J. W. Jobling. **1916** Richard Weil. **1917** J. A. Kolmer. **1918** W. H. Park. **1919** Hans Zinsser. **1920** R. I. Cole. **1921** F. P. Gay. **1922** G. W. McCoy. **1923** H. G. Wells. **1924** F. G. Novy. **1925** W. H. Manwaring. **1926** Ludvig Hektoen. **1927** Karl Landsteiner. **1928** E. L. Opie. **1929** O. T. Avery. **1930** Stanhope Bayne-Jones. **1931** A. R. Dochez. **1932** A. B. Wadsworth. **1933** T. M. Rivers. **1934** F. G. Blake. **1935** W. T. Longcope. **1936** S. B. Hooker. **1937** Carl TenBroeck. **1938** D. T. Fraser. **1939** G. P. Berry. **1940** P. R. Cannon. **1941** K. F. Meyer. **1942–1945** J. J. Bronfenbrenner. **1945–1947** Michael Heidelberger. **1947** L. D. Felton. **1948** Michael Heidelberger. **1949** Thomas Francis, Jr. **1950** Geoffrey Edsall. **1951** C. M. MacLeod. **1952** J. F. Enders. **1953** T. P. Magill. **1954** A. M. Pappenheimer, Jr. **1955** Jules Freund. **1956** M. W. Chase. **1957** J. H. Dingle.

Vice-Presidents–**1913–1915** G. W. Ross. **1915** G. P. Sanborn. **1916** J. A. Kolmer. **1947** Michael Heidelberger. **1948** L. D. Felton. **1949** Michael Heidelberger. **1950** C. M. MacLeod. **1951** J. F. Enders. **1952** T. P. Magill. **1953** A. M. Pappenheimer, Jr. **1954** Jules Freund. **1955** M. W. Chase. **1956** J. H. Dingle. **1957** J. E. Smadel.

Secretary–**1913–1918** M. J. Synott. **1946–1947** A. F. Coca.

Treasurer–**1913–1918** W. J. Stone. **1946–1947** A. J. Weil.

Secretary-Treasurer–**1918–1945** A. F. Coca. **1948–1950** Jules Freund. **1951–1953** J. Y. Sugg. **1954–1958** F. S. Cheever.

CONSTITUTION

(As revised, 1949)

ARTICLE I. *Name*

This association shall be called THE AMERICAN ASSOCIATION OF IMMUNOLOGISTS.

ARTICLE II. *Object*

The purpose of the Association shall be to advance knowledge of immunology and related disciplines, and to facilitate interchange of ideas and information among investigators in the various fields.

ARTICLE III. *Members*

SECTION 1. The Association shall consist of active members, members emeriti and honorary members.

SECTION 2. Any qualified person engaged in the study of problems related to the purpose of the Association may apply for active membership. Candidates for active membership shall be nominated by two members of the Association on blanks furnished by the Secretary. Applications must be accompanied by letters of recommendation of the sponsors, a curriculum vitae, and a list of reprints of publications. The Council shall determine eligibility and post a list of candidates at the annual meeting. The membership shall elect new members by majority vote.

SECTION 3. Failure to pay dues for three successive years shall annul membership. The Council may reinstate a member if an acceptable explanation is submitted and all indebtedness to the Association is liquidated. Payment of such indebtedness may be waived by unani-

mous vote of the Council if circumstances justify such action.

SECTION 4. If a two-thirds majority of the Council decides that the best interests of the Association require the expulsionof a member, the Secretary shall notify the affected member in writing of the charges. The Council shall allow a reasonable time for the presentation of his defense before acting. Upon recommendation of a two-thirds majority of the Council, the Secretary shall send a notice of the decision to each active member at least six weeks before the next annual meeting. At this meeting the Secretary shall, on behalf of the Council, propose the expulsion; and on a two-thirds vote of the members present, the member shall be expelled, his assessment for the current year shall be returned, and he shall cease to be a member of the Association.

SECTION 5. An active member of the Association for twenty years upon retirement because of age or disability, may elect to accept the status of member emeritus. As such he shall retain voting privileges but shall be relieved of all financial obligations to the Association and shall not receive the Journal without payment.

SECTION 6. The Council may nominate for honorary membership persons of extraordinary achievement in the field of endeavor of this Association. Election to honorary membership shall follow the same procedure as that for election for office in the Association, and the Secretary, on order of the Council, shall place nominations for honorary membership on the annual ballot.

ARTICLE IV. *Officers*

SECTION 1. The Association shall be governed by a Council, which shall consist of the Officers of the Association and five Councilors, one of whom shall be the Editor-in-Chief of the Journal of Immunology.

SECTION 2. The Officers of the Association shall be a President, a Vice President, a Secretary, and a Treasurer. The duties of the two latter may be performed by one or two individuals as determined by vote of the Association. (See By-Law # 7.)

SECTION 3. The President, the Vice President, the Secretary, and the Treasurer shall be elected at the regular annual meeting of the Association to serve for one year. Only the Secretary and the Treasurer are eligible to serve for successive terms in their respective offices. The term of office shall begin the day after the end of the annual meeting.

SECTION 4. One Councilor shall be elected each year to serve for a term of four years. He may however serve in any other elective office immediately after expiration of his term as Councilor.

SECTION 5. The President shall appoint a Nominating Committee of three (or more) members not currently serving on the Council of the Association and shall designate the Chairman. The Committee shall submit nominations for the offices of President, Vice President, Secretary, Treasurer, and Councilor. They may nominate the member currently serving as Vice President for the office of President, and the Councilor who is serving his fourth year for the office of Vice President. They shall submit the names of three members of the Association as candidates for the office of Councilor for a four-year term. The Nominating Committee shall confer at least three months before the annual meeting and shall transmit its nominations, over the signatures of the majority of the Committee, to the Secretary at least two months before the annual meeting. Other names may be added to the ballot upon petition of at least twenty-five members of the Association to the Secretary at least two months before the annual meeting. The Secretary shall send to each member of the Association, at least six weeks before the annual meeting, a ballot containing the list of all such nominees.

SECTION 6. The members of the Association shall vote on the nominations by secret ballot by mail. All ballots must be in the hands of the Secretary one week before the annual meeting. At the time of the annual meeting the Secretary shall present all the valid sealed ballots received by him to the tellers appointed by the President. A plurality of votes shall be sufficient for election.

SECTION 7. The Vice President shall substitute for the President when necessary. If both the President and Vice President are unable to serve, the senior Councilor shall temporarily assume the duties of President. Should a vacancy occur in the office of Secretary or Treasurer, or Councilor, the Council by a majority vote shall appoint a member to fill the unexpired term until the following election. In the event of a vacancy in the Council, the members shall in the following election choose two Councilors from among six nominees whose names shall be selected by the Nominating Committee and included on the ballot as above. Moreover, in this event the terms of the four Councilors shall be adjusted so as to provide an orderly progression within the general sense of the Constitution.

ARTICLE V. *Meetings*

SECTION 1. A regular meeting of the Association shall be held annually at such time and place as the Council shall determine. Regular meetings shall be open to all members of the Association. The members present at the annual meeting of the Association shall constitute a quorum.

SECTION 2. A meeting of the Council shall be held prior to the annual business meeting of the Association. A quorum of the Council for this meeting shall be five.

SECTION 3. In case of equal division of votes, the President shall cast the decisive ballot.

SECTION 4. Minutes of the annual meeting

of the Council and of the annual business meeting of the Association shall be published in the form of a news-letter and be distributed only to members of the Association. The annual report of the Treasurer shall be included in this newsletter.

ARTICLE VI. *Business*

SECTION 1. The fiscal year of the Association shall begin March first.

SECTION 2. Annual dues, upon recommendation of the Council, shall be determined by a majority vote at the annual meeting of the Association.

ARTICLE VII. *Publication*

SECTION 1. The Journal of Immunology, which is the property and official organ of this Association, shall be administered for the Association by an Editorial Board.

SECTION 2. An Editor-in-Chief shall be elected from the membership by a majority vote of the Council to serve for four years. His term of office shall be subject to renewal. He shall appoint with the consent of the Council an Editorial Board of four to six members to serve for four years. The Editor-in-Chief and the members of the Editorial Board shall be responsible for the editorial conduct of the Journal.

SECTION 3. The Editor-in-Chief shall, with the consent of the Council, appoint from the active members Associate Editors as needed. These Associate Editors are to be chosen because of their ability in specialized fields.

SECTION 4. The Editor-in-Chief shall make a report of his stewardship of the Journal at the annual meeting and in this report shall summarize the editorial situation and all matters dealing with finances. He shall submit to the Council statements relating to the number of manuscripts received, rejected, accepted, and published during the year; changes in editorial personnel of editors; a complete summary of circulation and of finances and of any other information which the Editorial Board may feel to be pertinent or which may be required by the Council.

ARTICLE VIII. *Amendments*

SECTION 1. Proposed changes in the Constitution shall be submitted by at least five members in writing through the Secretary to the President. The President shall then appoint a committee of at least three members which shall communicate its recommendations to the President for consideration by the Council. The Council shall then advise the Secretary to submit the recommendations of the committee to the membership of the Association for approval with the annual ballot. A change in the Constitution shall require a two-thirds majority of the votes cast by the members at the annual meeting and shall require confirmation by a majority in a mail vote in which at least 50% shall have participated.

SECTION 2. As an alternative procedure a proposed change in the Constitution may be submitted directly to the Council by petition of one-tenth of the total active membership. Under this circumstance the Council is required to arrange for a vote by the members as heretofore described.

SECTION 3. Proposed changes in By-Laws may be adopted by two-thirds vote of members present at the annual meeting.

BY-LAWS

1. The Past Presidents shall have the right of attending, without vote, the meetings of the Council.

2. The President may appoint a Past President or a Past Councilor as pro tempore Councilor at any stated meeting of the Council at which a quorum is not present.

3. The Council may transact and vote by mail on such business as cannot be conveniently transacted at meetings.

4. The Secretary shall arrange the program for the scientific meetings, with the advice of the other officers of the Association. Any member in good standing shall have the right to submit a paper. The privilege of presentation may be extended to non-members upon recommendation of active members. Papers intended for presentation at the meetings shall conform to the standards of the Journal of Immunology. In case of doubt, the Secretary shall have the right to submit papers to the scrutiny of three or more members of the Editorial Board of the Journal of Immunology whose decision shall be final.

5. Each member who signifies his intention of reading a paper at any session may specify the length of time which its presentation shall require. In the absence of any specification of time required not more than ten minutes shall be allotted for the reading of any one paper. The time alloted shall be printed on the official program, and the presiding officer shall have no authority to extend it unless a majority of the members present signify their wish to the contrary.

6. If by force of circumstances it should be impossible to hold the annual meeting, the election of Officers and Council may be carried out entirely by mail.

7. The offices of Secretary and Treasurer are combined and shall be held by one person.

8. Official or invited addresses presented at the annual meeting shall be given immediate priority in publication, unless the Editor-in-Chief shall be otherwise instructed by majority vote of the Council.

9. Dues are payable on or before January 1st and subscriptions to the Journal will be lapsed if not paid by this date.

10. If the Editor-in-Chief is unable to attend the stated meeting of the Council, he may designate an alternate from the membership of the Editorial Board.

ALPHABETICAL LIST OF MEMBERS OF THE SIX SOCIETIES[1]

(1) The American Physiological Society
(2) The American Society of Biological Chemists
(3) The American Society for Pharmacology and Experimental Therapeutics
(4) The American Society for Experimental Pathology
(5) The American Institute of Nutrition
(6) The American Association of Immunologists

Number and year in parentheses following each name indicate Society affiliation and year of election
H signifies honorary member; R signifies retired status

AAES–JORGENSEN, ERIK, Ph.D. Polytechnic Inst., Dept. of Biochem. and Nutrition, Copenhagen, Denmark. *Lect.* (5, 1958)

ABBOTT, LYNN D. F., JR., Ph.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Biochem.* (2, 1948)

ABELL, LIESE L., Ph.D. Columbia U. Res. Serv., Goldwater Mem. Hosp., Welfare Is., New York 17. *Asst. Prof., Biochem., Columbia U.* (2, 1957)

ABOOD, LEO G., Ph.D. U. of Illinois Coll. of Med., Dept. Psychiatry, Chicago 12. *Assoc. Prof. Biochem. and Neurophysiol.* (1, 1951)

ABRAHAM, SAMUEL, Ph.D. U. of California Sch. of Med., Berkeley 4. *Asst. Research Physiologist.* (2, 1956)

ABRAMS, ADOLPH, Ph.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Biochem.* (2, 1955)

ABRAMS, RICHARD, Ph.D. Montefiore Hosp., Inst. of Research, Pittsburgh 13, Pa. *Dir.* (2, 1953)

ABRAMSON, DAVID I., M.D. U. of Illinois, Dept. of Physical Med., Chicago 12. *Prof. and Head of Dept.* (1, 1937)

ABRAMSON, HAROLD A., M.D. Biological Lab., Cold Spring Harbor, N.Y. *Research Psychiatrist.* (1, 1930; 2, 1934)

ABREU, BENEDICT E., Ph.D., M.D. Pitman-Moore Co., Indianapolis 6, Ind. *Assoc. Research Dir.* (3, 1941)

ACHESON, GEORGE H., M.D. U. of Cincinnati Coll. of Med., Cincinnati 19, Ohio. *Prof. of Pharmacol.* (3, 1945)

ACHOR, LEONARD B., Ph.D., M.D. Merck Inst. for Therap. Research, Rahway, N.J. *Research Assoc.* (3, 1957)

ACKERMANN, W. WILBUR, Ph.D. 116 N. State St., Ann Arbor, Mich. *Assoc. Prof. of Epidemiol., U. of Michigan.* (2, 1952; 6, 1953)

ADAMS, ELIJAH, M.D. St. Louis U. Sch. of Med., Dept. of Pharmacol., St. Louis, Mo. *Prof. and Dir.* (2, 1954)

ADAMS, GEORGIAN, D.Sc. USDA, State Expt. Stations Div., ARS, Washington 25, D.C. *Agric. Admin.* (5, 1946)

ADAMS, JOHN M., M.D., Ph.D. U. of California, 405 Hilgard Ave., Los Angeles 24. *Prof. and Chmn. of Pediatrics.* (4, 1947)

ADAMS, MILDRED, Ph.D. USDA, Human Nutrition Research Br., Beltsville, Md. *Head, Nutritional Requirements Sec.* (2, 1934)

ADAMS, W. LLOYD, M.D., Ph.D. 900 First Natl. Bank Bldg., Lexington 6, Ky. *Practicing Ophthalmologist.* (3, 1942)

ADAMS, WRIGHT R., M.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Prof. and Chmn. of Dept.* (1, 1946)

ADDISON, WILLIAM H., M.D. U. of Pennsylvania, Philadelphia. *Prof. Emer. of Histol. and Embryol.* (1R, 1928)

ADES, HARLOW WHITING, Ph.D. USN Sch. of Aviation Med., Pensacola NAS, Fla. *Head, Dept. Neurophysiol. & Acoustics.* (1, 1953)

ADEY, W. R., M.D. U. of California Med. Ctr., Los Angeles 24. (1, 1955)

ADLER, FRANK L., Ph.D. Public Health Research Inst. of City of New York, Foot of E. 15th St., New York City 9. *Assoc. Member.* (6, 1953)

ADLER, HARRY F., Ph.D., M.D. 274 W. Ware Blvd., San Antonio 24, Tex. (1, 1943)

ADLER, TERRINE K., Ph.D. U. of California Med. Ctr., San Francisco 22. *Asst. Research Pharmacologist.* (3, 1954)

ADOLPH, EDWARD FREDERICK, Ph.D. U. of Rochester Med. Ctr., Rochester 20, N.Y. *Prof. of Physiol.* (1, 1921)

ADOLPH, WILLIAM H., Ph.D. Yale U., Sterling Hall of Med., New Haven, Conn. (2R, 1946; 5, 1934)

ADRIAN, E. D. Cambridge U., Trinity Coll., Cambridge, England. (1H, 1946)

AGRESS, CLARENCE M., M.D. U. of California, Los Angeles. *Assoc. Clin. Prof. of Med.* (1, 1955)

AHLQUIST, RAYMOND P., Ph.D. Med. Coll. of Georgia, Dept. of Pharmacol., Augusta. *Prof. and Chmn. of Dept.* (1, 1952; 3, 1945)

AHRENS, EDWARD H., JR., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (2, 1956)

AJAZZI–MANCINI, MARIO, M.D. U. of Firenze, Dept. of Pharmacol., Viale G. B. Morgagni 65, Firenze, Italy. *Dir. of Pharmacol.* (3, 1951)

[1] *Includes changes received up to July 15, 1958.*

AJMONE-MARSAN, COSIMO, M.D. Natl. Inst. of Neurological Diseases & Blindness, NIH, Bethesda 14, Md. *Chief, Br. of EEG.* (1, 1956)

AKERS, ROBERT P., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Research Physiologist.* (1, 1955)

AKERT, KONRAD, M.D. U. of Wisconsin, Serv. Mem. Insts., Madison. *Assoc. Prof. of Physiol.* (1, 1957)

ALADJEM, FREDERICK, Ph.D. U. of Southern California Sch. of Med., Los Angeles 7. *Asst. Prof. of Med. Microbiol.* (6, 1957)

ALBANESE, ANTHONY A., Ph.D. St. Luke's Convalescent Hosp., Nutritional Research Lab., Greenwich, Conn. *Dir. of Research.* (2, 1944)

ALBAUM, HARRY G., Ph.D. Brooklyn Coll., Brooklyn 10, N.Y. *Prof. of Biol.* (2, 1947)

ALBERT, A., Ph.D., M.D. Mayo Clinic, Rochester, Minn. *Head, Endocrinol. Lab.; Prof. of Physiol., Mayo Fndn.* (1, 1947)

ALBERTY, ROBERT A., Ph.D. U. of Wisconsin, Dept. of Chem., Madison 6. *Prof.* (2, 1956)

ALBRITTON, ERRETT C., M.D. Div. of Research Grants, NIH, Bethesda 14, Md. *Spec. Asst. to Sci. Dir.* (1R, 1933)

ALDEN, ROLAND H., Ph.D. U. of Tennessee, Med. Units, Div. of Anatomy, Memphis 3. *Prof. and Chief of Div.* (1, 1949)

ALDERTON, GORDON, M.S. USDA, Western Regional Research Lab., Albany 10, Calif. *Chemist.* (2, 1952)

ALEXANDER, BENJAMIN, M.D. Beth Israel Hosp., Boston, Mass. *Assoc. Dir., Med. Serv.; Assoc. Prof. of Med., Harvard Med. Sch.* (2, 1951)

ALEXANDER, NATALIE, Ph.D. Kerckhoff Labs., Los Angeles 7, Calif. *Asst. Prof. Dept. of Physiol., U. of Southern California.* (1, 1958)

ALEXANDER, ROBERT S., Ph.D. Albany Med. Coll. Dept. of Physiol., Albany 8, N.Y. *Prof. and Chmn. of Dept.* (1, 1946)

ALFIN–SLATER, ROSLYN B., Ph.D. U. of Southern California, Dept. of Biochem. & Nutr., Los Angeles 7. *Visiting Assoc. Prof.* (2, 1952; 5, 1955)

ALFREDSON, B. V., D.V.M. Michigan State U., Dept. of Physiol. and Pharmacol., East Lansing. *Prof. and Head of Dept.* (1, 1951)

ALIVISATOS, S. G. A., M.D., Ph.D. Mt. Sinai Med. Res. Fndn., Dept. Biochem. Res., Chicago 8, Ill. *Head; Assoc., Dept. Pathol., Chicago Med. Sch.* (2, 1958)

ALLAN, FRANK N., M.D. Lahey Clinic, 605 Commonwealth Ave., Boston, Mass. *Exec. Dir. of Med. Dept.* (4, 1930)

ALLEN, CHARLES ROBERT, Ph.D., M.D. U. of Texas Sch. of Med., Dept. of Anesthesiol., Galveston. *Prof. and Chmn.* (1, 1943)

ALLEN, FRANK W., Ph.D. U. of California Sch. of Med., 960 Med. Sci. Bldg., San Francisco. *Prof. of Biochem.* (2, 1947)

ALLEN, FREDERICK M., M.D. 1031 Fifth Ave., New York City 28. (1R, 1924; 4, prior to 1920)

ALLEN, J. GARROTT, M.D. U. of Chicago Clinics, Chicago 37, Ill. *Prof. of Surgery.* (1, 1943)

ALLEN, LANE, Ph.D., M.D. Med. Coll. of Georgia, University Place, Augusta. *Prof. of Gross Anatomy.* (1, 1939)

ALLEN, SHANNON C., Ph.D. Chicago Coll. of Osteopathy, Dept. of Physiol., Chicago 15, Ill. *Asst. Prof.* (1, 1945)

ALLEN, THOMAS H., Ph.D. USA Med. Nutrition Lab., Fitzsimons Army Hosp., Denver 8, Colo. *Chief, Physiol. Div.* (1,1947)

ALLEN, WILLARD M., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Obstetrics and Gynecol.* (1, 1934)

ALLES, GORDON A., Ph.D. 770 S. Arroyo Pkwy., Pasadena 2, Calif. *Prof. of Pharmacol. in Res., UCLA Sch. of Med.* (1, 1932; 3, 1941)

ALLING, ERIC L., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Assoc. in Med.; Assoc. in Radiation-Biol.* (4, 1947)

ALLISON, JAMES B., Ph.D. Rutgers, The State U., New Brunswick, N.J. *Prof. of Physiol. & Biochem.; Dir., Bur. of Biol. Res.* (2, 1946; 5, 1949)

ALMQUIST, HERMAN J., Ph.D., D.Sc. The Grange Co., Modesto, Calif. *Dir. of Research and Vice-Pres.* (2, 1937; 5, 1937)

ALPEN, EDWARD L., Ph.D. U.S. Naval Radiol. Defense Lab., Div. Biol. & Med. Sci., San Francisco 24, Calif. *Head, Biophysics Br.* (1, 1955)

ALTAMIRANO, MARIO, M.D. Universidad de Chile, Instituto de Fisiologia, Santiago, Chile. *Asst. Prof. of Physiol.* (1, 1955)

ALTLAND, PAUL D., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Physiologist.* (1, 1950)

ALTMAN, KURT I., M.D. U. of Rochester Sch. of Med. and Dent., Dept. Radiation Biol., Rochester 20, N.Y. *Scientist, AEC; Assoc. in Radiol.* (2, 1958)

ALTSCHUL, AARON M., Ph.D. USDA, Box 7307, New Orleans 19, La. *Prin. Chem., Seed Protein Pioneering Res. Lab.* (2, 1949)

ALTSHULER, CHARLES H., M.D. St. Joseph's Hosp., Milwaukee, Wis. *Pathologist.* (4, 1956)

ALTSZULER, NORMAN, Ph.D. New York U. Coll. of Med., Dept. of Pharmacol., New York City 16. *Asst. Prof.* (1, 1958)

ALVAREZ, WALTER C., M.D. 700 N. Michigan Ave., Chicago 11, Ill. (1R, 1917; 3R, 1921)

ALVING, ALF SVEN, M.D. Billings Hosp., U. of Chicago, Chicago 37, Ill. *Prof. of Med.* (1, 1939)

ALVORD, ELLSWORTH C., JR., M.D. Baylor U. Coll. of Med., Houston, Tex. *Assoc. Prof. of Neurol. and Pathol.* (4, 1956)

AMASSIAN, VAHE EUGENE, M.B., B.Ch. Albert Einstein Coll. of Med., New York City 61. *Prof. of Physiol.* (1, 1953)

AMBERG, SAMUEL, M.D. Mayo Clinic, Rochester, Minn. *Assoc. Prof. Emer. of Pediatrics, Mayo Fndn.* (1R, 1903; 2R, 1906; 3R, 1909)

AMBERSON, WILLIAM R., Ph.D. U. of Maryland Sch. of Med., Baltimore 1. *Prof. of Physiol.* (1, 1924)

AMBROSE, ANTHONY M., Ph.D. USA Environ. Health Lab., Army Med. Serv., Army Chemical Center, Md. *Chief, Toxicol. Div.* (3, 1937)

AMBRUS, CLARA M., M.D., Ph.D. Roswell Pk. Mem. Inst., Buffalo, N.Y. *Asst. Prof. of Pharmacol., U. of Buffalo.* (1, 1957; 3, 1953)

AMBRUS, JULIAN L., M.D., Ph.D. Roswell Pk. Mem. Inst., Buffalo, N.Y. *Prin. Cancer Res. Scientist, State of New York.* (1, 1957; 3, 1953)

AMES, BRUCE N., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Biochemist.* (2, 1957)

AMES, STANLEY R., Ph.D. Distillation Products Ind., Div. of Eastman Kodak Co., Rochester 3, N.Y. *Research Assoc.* (2, 1948)

ANCHEL, MARJORIE, Ph.D. New York Botanical Garden, Bronx Park, New York City 58. *Research Assoc.* (2, 1951)

ANDERSCH, MARIE A., Ph.D. University Hosp., Baltimore 1, Md. *Asst. Dir. of Lab.; Assoc. Prof. of Biochem. in Med.* (2, 1940)

ANDERSEN, DOROTHY H., M.D. Presbyterian Hosp., New York City 32. *Prof. of Patbol., Columbia U.* (4, 1935)

ANDERSON, CARL E., Ph.D. U. of North Carolina, Sch. of Med., Chapel Hill. *Assoc. Prof. of Biochem. & Asst. Dean.* (2, 1950)

ANDERSON, ELIZABETH P., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chemist, Leukemia Studies Sec.* (2, 1957)

ANDERSON, EVELYN, M.D., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Sec. of Endocrinol.* (1, 1934)

ANDERSON, HAMILTON H., M.D. U. of California Sch. of Med., Dept. of Pharmacol. and Exptl. Therap., San Francisco 22. *Prof.* (3, 1931)

ANDERSON, JOSEPH T., Ph.D. U. of Minnesota, Lab. of Physiol. Hygiene, Minneapolis 14. *Assoc. Prof.* (5, 1952)

ANDERSON, LAURENS, Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Assoc. Prof.* (2, 1953)

ANDERSON, NORMAN G., Ph.D. Oak Ridge Natl. Lab., Biology Div., Oak Ridge, Tenn. *Biologist.* (1, 1954)

ANDERSON, ROBERT C., B.S. Lilly Research Labs., Eli Lilly & Co., Indianapolis 6, Ind. *Head, Dept. of Toxicol.* (3, 1954)

ANDERSON, RUBERT S., Ph.D. Chem. Warfare Labs., Directorate of Med. Research, Army Chemical Center, Md. (1, 1948)

ANDERSON, RUDOLPH J., Ph.D. 101 Cottage St., New Haven, Conn. *Prof. of Chem.* (2, 1915)

ANDERSON, W. A. D., M.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Prof. of Patbol.* (4, 1941)

ANDERSON, WILLIAM E., M.A. Tolland St., Tolland, Conn. (2R, 1931; 5, 1933)

ANDERVONT, H. B., D.Sc. Natl. Cancer Inst. NIH, Bethesda 14, Md. *Chief, Lab. of Biol.* (4, 1939)

ANDRES, REUBIN, M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Asst. Prof., Environ. Med.* (1, 1955)

ANDRESEN, RICHARD H., M.D. Presbyterian-St. Luke's Hosp., Chicago 12, Ill. *Research Assoc. in Patbol.* (4, 1957)

ANDREWS, JAMES C., Ph.D. Box 392, Conway, N.H. *Emer. Prof. of Biochem., U. of North Carolina Sch. of Med.* (2R, 1925)

ANDRUS, E. COWLES, M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. of Med., Johns Hopkins U.* (1, 1925)

ANDRUS, STEPHEN B., M.D. Harvard Sch. of Public Health, Dept. of Nutrition, Boston 15, Mass. *Asst. Prof. of Patbol.* (4, 1956)

ANFINSEN, CHRISTIAN B., JR., Ph.D. Natl. Heart Inst., Lab. of Cellular Physiol. and Metab., NIH, Bethesda 14, Md. *Lab. Chief.* (2, 1946)

ANGERER, CLIFFORD A., Ph.D. Ohio State U. Coll. of Med., Dept. of Physiol., Columbus 10. *Prof.* (1, 1943)

ANGEVINE, D. MURRAY, M.D. U. of Wisconsin Med. Sch., Madison 6. *Prof. of Patbol.* (4, 1940)

ANKER, H.S., M.D., Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Assoc. Prof.* (2, 1949)

ANNEGERS, JOHN H., Ph.D., M.D. Northwestern U. Med. Sch., Dept. of Physiol., Chicago 11, Ill. *Assoc. Prof.* (1, 1949)

ANSBACHER, STEFAN, Sc.D. Jocinah Farms, R.R. 1, Marion, Ind. *Sci. and Med. Consultant.* (2, 1939)

ANSLOW, W. PARKER, JR., Ph.D. U. of Virginia Sch. of Med., Dept. of Physiol., Charlottesville. *Prof. and Chmn. of Dept.* (1, 1955; 2, 1948)

ANSON, MORTIMER L., Ph.D. 100 Eaton Sq., London S.W. 1, England. (2, 1937)

ANTOPOL, WILLIAM, M.D. Beth Israel Hosp., 10 Livingstone Pl., New York City 3. *Dir. Research, Joseph H. Levy Fndn.* (4, 1953)

APOSHIAN, H. VASKEN, Ph.D. Vanderbilt U. Sch. of Med., Nashville 5, Tenn. *Asst. Prof.* (3, 1958)

APPERLY, FRANK L., D.Sc., M.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Patbol.* (4, 1936)

APRISON, MORRIS H., Ph.D. Indiana U. Med. Ctr., Inst. of Psychiatric Res., Indianapolis. *Asst. Prof. of Biochem.* (1, 1955)

APTEKMAN, PAUL MYRON, Ph.D. The Wistar Inst., Philadelphia 4, Pa. *Curator of Museum; Fellow in Chem.* (6, 1953)

ARCHDEACON, JAMES W., Ph.D. U. of Kentucky, Dept. of Anatomy and Physiol., Lexington. *Prof.* (1, 1955)

ARCHIBALD, REGINALD M., Ph.D., M.D. Rockefeller Hosp., New York City 21. *Member & Prof., Rockefeller Inst. for Med. Res.* (2, 1947)

ARKIN, AARON, M.D., Ph.D. 25 E. Washington St., Chicago 2, Ill. *Rush Prof. Med., U. of Ill.; Prof. Med., Cook Co. Grad. Sch.* (1R, 1914; 3R, 1919)

ARMSTRONG, HARRY G., M.D. 4701 Connecticut Ave., N.W., Washington 8, D.C. *Maj. Gen., USAF, MC.* (1R, 1948)

ARMSTRONG, MARVIN D., Ph.D. Fels Research Inst., Dept. of Biochem., Yellow Springs, Ohio. *Chmn.* (2, 1951)

ARMSTRONG, PHILIP B., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Prof. of Anatomy.* (1, 1945)

ARMSTRONG, W. D., M.D., Ph.D. U. of Minnesota Med. Sch., Dept. of Physiol. Chem., Minneapolis 14. *Prof. and Head.* (1, 1955; 2, 1938)

ARNOLD, AARON, Ph.D. Sterling-Winthrop Research Inst., Rensselaer, N.Y. *Head of Nutritional Research Lab.* (5, 1947)

ARNOLD, JAMES S., M.D. VA Hosp., Dept. of Pathol., San Francisco, Calif. *Clin. Invest.* (4, 1956)

ARNOLD, LLOYD, M.D. 1538 E. 57th St., Chicago 37, Ill. (4, 1930; 6, 1925)

ARNON, DANIEL I., Ph.D. U. of California, 3044 Life Sci. Bldg., Berkeley 4. *Prof. of Plant Physiol.* (2, 1958)

ARNOW, L. EARLE, Ph.D., M.D. Warner-Lambert Research Inst., Morris Plains, N.J. *Pres.*

(2, 1940)

ARNRICH, LOTTE, Ph.D. Iowa State Coll., Ames. *Assoc. Prof. of Food and Nutrition.* (5, 1957)

ARONOFF, S., Ph.D. Iowa State Coll., Botany Hall, Ames. *Prof.* (2, 1954)

ARONSON, JOSEPH D., M.D. U. of Pennsylvania, Henry Phipps Inst., Philadelphia 4. *Prof. of Bacteriol.* (4, 1927; 6, 1925)

ARQUILLA, EDWARD R., Ph.D., M.D. Western Reserve U. Inst. of Pathol., Cleveland 6, Ohio. *Research Fellow.* (6, 1958)

ARROYAVE, GUILLERMO, Ph.D. Instituto de Nutricion de Centro America y Panama, Guatemala. *Chief, Div. of Clin. Biochem.* (5, 1958)

ARTOM, CAMILLO, M.D., Ph.D. Wake Forest Coll., Bowman Gray Sch. of Med., Winston-Salem, N. C. *Prof. of Biochem.* (2, 1944)

ARTUSIO, JOSEPH F., JR., M.D. Cornell U. Med. Coll., Dept. of Surgery, New York City 21. *Prof., Anesthesiol. in Surg.* (3, 1956)

ASCHAM, LEAH, Ph.D. Kansas State Coll., Manhattan. *Prof. Emer. in Sch. of Home Economics.* (5R, 1935)

ASENJO, CONRADO F., Ph.D. U. of Puerto Rico Sch. of Med., Dept. of Biochem. and Nutrition, San Juan 22. *Prof. and Head.* (2, 1944; 5, 1951)

ASHBY, WINIFRED M., Ph.D. 305-10th St., N. E., Washington 2, D. C. (6, 1923)

ASHLEY, CHARLES A., M.D. Mary Imogene Bassett Hosp., Cooperstown, N. Y. *Assoc. Pathologist; Instr. in Pathol., Columbia U.* (4, 1956)

ASHMAN, RICHARD, Ph.D. 5902 Marshal Foch St., New Orleans, La. *Prof. Emer. of Physiol., Louisiana State U.* (1R, 1925)

ASHMORE, JAMES, Ph.D. Indiana U., Biochem. Dept., Indianapolis. *Assoc. Prof.* (2, 1956)

ASHWELL, G. GILBERT, M.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Surgeon, PHS.* (2, 1956)

ASTWOOD, EDWIN BENNETT, M.D., Ph.D. New England Ctr. Hosp., Boston 11, Mass. *Prof. of Med., Tufts U.* (1, 1939)

ATKIN, LAWRENCE, Ph.D. Fleischmann Labs., Standard Brands, Inc., Betts Ave., Stamford, Conn. *Asst. Dir. of Research.* (2, 1946; 5, 1946)

ATKINSON, DANIEL E., Ph.D. U. of California, Dept. of Chem., Los Angeles 24. *Assoc. Prof.* (2, 1957)

AUB, JOSEPH C., M.D. Massachusetts Gen. Hosp., Boston 14. *Emer. Prof. of Res. Med., Harvard Med. Sch.* (1R, 1919; 5, 1933)

AUERBACH, STEWART H. Baroness Erlanger Hosp., Chattanooga, Tenn. (4, 1953)

AUSTIN, GEORGE M., M.D. U. of Oregon Med. Sch., Div. of Neurosurgery, Portland 1. *Prof. and Head of Div.* (1, 1954)

AVIADO, DOMINGO M., JR., M.D. U. of Pennsylvania Sch. of Med., Dept. of Pharmacol., Philadelphia 4. *Asst. Prof.* (1, 1951; 3, 1950)

AWAPARA, JORGE, Ph.D. The Rice Inst., Houston, Tex. *Assoc. Prof. of Biochem.* (2, 1951)

AXELROD, ABE E., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Biochem., Pittsburgh 13, Pa. *Prof.* (2, 1950; 5, 1949; 6, 1956)

AXELROD, BERNARD, Ph.D. Purdue U., Dept. of Biochem., Lafayette, Ind. *Prof.* (2, 1948)

AXELROD, D. ROBERT, M.D. Cornell U. Med. Coll., New York 21. *Asst. Prof. of Physiol.; Chief, Metabolic Res. Unit, Brooklyn VA Hosp.* (1, 1958)

AXELROD, JULIUS, Ph.D. Natl. Inst. of Mental Health, Lab. of Clin. Sci., NIH, Bethesda 14, Md. *Chief, Sec. on Pharmacol.* (3, 1953)

BACCHUS, HABEEB, Ph.D., M.D. George Washington U. Sch. of Med., Dept. of Physiol., Washington 5, D. C. *Asst. Res. Prof.* (1, 1952)

BACH, L. M. N., Ph.D. Tulane U. Sch. of Med., New Orleans, La. *Prof. of Physiol.* (1, 1948)

BACHER, JAMES E., Ph.D. 3759 Woodruff Ave., Oakland 2, Calif. (2, 1953)

BACHMAN, CARL, M.D. U. of Pennsylvania, Philadelphia 4. *Prof. of Obstetrics & Gynecol.* (2, 1941)

BACHMANN, GEORGE, M.D. 1088 Lullwater Rd., N. E., Atlanta 7, Ga. *Prof. Emer. of Physiol., Emory U. Sch. of Med.* (1R, 1912)

BACHRACH, WILLIAM H., M.D., Ph.D. U. of Southern California Sch. of Med., Los Angeles 7. *Assoc. Clin. Prof. of Med.* (1, 1949)

BACK, NATHAN, D.Sc. Roswell Park Mem. Inst., Sec. of Physiol. & Pharmacol., Buffalo 3, N. Y. *Cancer Research Scientist.* (3, 1958)

BAEDER, DAVID H., Ph.D. Wyeth Inst. for Med. Research, Pharmacol. Sec., Philadelphia 1, Pa. *Sr. Research Pharmacologist.* (3, 1956)

BAER, ERICH, Ph.D. U. of Toronto, Banting & Best Dept. of Med. Res., Toronto, Canada. *Prof.* (2, 1942)

BAER, HAROLD, Ph.D. Tulane U. Sch. of Med., Dept. of Microbiol., New Orleans, La. *Assoc. Prof.* (6, 1955)

BAERNSTEIN, HARRY D., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Biochemist.* (2, 1934)

BAETJER, ANNA M., D.Sc. Johns Hopkins U. Sch. of Hygiene & Public Health, Baltimore 5, Md. *Assoc. Prof. Environ. Med.* (1, 1929)

BAEZ, SILVIO, M.D. New York U.-Bellevue Med. Ctr., New York City 16. *Asst. Prof.* (1, 1950)

BAGGETT, BILLY, Ph.D. U. of North Carolina Sch. of Med., Dept. of Pharmacol., Chapel Hill. *Asst. Prof.* (2, 1957)

BAHN, ROBERT C., M.D., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant, Sec. Patbol. Anatomy.* (1, 1955)

BAILEY, CAMERON VERNON, C.M., M.D. 26 West St., Newtown, Conn. (2R, 1920; 5R, 1933)

BAILEY, ORVILLE T., M.D. Larue D. Carter Mem. Hosp., Indianapolis 7, Ind. *Prof. of Neuropathol., Indiana U. Sch. of Med.* (4, 1939)

BAILEY, PERCIVAL, M.D., Ph.D. U. of Illinois Coll. of Med., Chicago 12, Ill. *Prof. of Neurol.* (1, 1941)

BAIN, JAMES A., Ph.D. Emory U., Div. Basic Health Sci., Dept. of Pharmacol., Atlanta 22, Ga. *Prof. and Chmn.* (3, 1951)

BAITSELL, GEORGE ALFRED, Ph.D. Yale U., Osborn Zool. Lab., Yale Sta., New Haven, Conn. *Emer. Colgate Prof. of Biol.* (1R, 1915)

BAKER, A. B., M.D. U. of Minnesota Hosp., Minneapolis 14. *Dir. and Prof. of Neurol. and Neuropathol.* (4, 1940)

BAKER, BURTON L., Ph.D. U. of Michigan, Dept. of Anatomy, Ann Arbor. *Prof.* (1, 1951)

BAKER, CARL G., M.D. Natl. Insts. of Health, Office of the Dir., Bethesda 14, Md. *Sr. Surgeon, PHS.* (2, 1955)

BAKER, EDGAR E., Ph.D. Boston U. Sch. of Med., Boston 18, Mass. *Prof. of Microbiol.* (6, 1950)

BAKER, JAMES A., D.V.M., Ph.D. Cornell U., New York State Veterinary Coll., Ithaca. *Dir., Veterinary Virus Research.* (4, 1947)

BAKER, NOME, Ph.D. VA Ctr., Radioisotope Serv., Los Angeles 25, Calif. *Prin. Scientist; Asst. Clin. Prof., U. of California Med. Ctr.* (2, 1957)

BAKER, ROGER D., M.D. VA Hosp., Durham, N.C. *Chief, Lab. Serv.; Prof. of Patbol., Duke U. Sch. of Med.* (4, 1939)

BAKER, WALTER W., Ph.D. Jefferson Med. Coll., Dept. of Pharmacol., Philadelphia 7, Pa. *Asst. Prof.* (3, 1955)

BALDES, EDWARD J., Ph.D. Mayo Clinic, Rochester, Minn. *Chmn., Sec. Biophysics & Biophys. Res.; Prof. Biophysics, Mayo Fndn. Grad. Sch.* (1, 1933)

BALDWIN, ROBERT L., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Asst. Prof.* (2, 1957)

BALE, WILLIAM F., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. of Radiation Biol.* (1, 1943)

BALIS, M. EARL, Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc. Member.* (2, 1953)

BALL, ERIC G., Ph.D. Harvard Med. Sch., 25 Shattuck St., Boston 15, Mass. *Prof. of Biol. Chem.* (2, 1934)

BALL, HOWARD A., M.D. 233 A St., San Diego 1, Calif. *Pathologist.* (4, 1947)

BALLOU, CLINTON E., Ph.D. U. of California, Dept. of Biochem., Berkeley. *Assoc. Prof.* (2, 1956)

BALLS, ARNOLD KENT, Ph.D. Purdue U., Dept. of Biochem., Lafayette, Ind. *Prof.* (2, 1932)

BANCROFT, RICHARD W., Ph.D. USAF Sch. of Aviation Med., Randolph AFB, Tex. *Physiologist.* (1, 1955)

BANDURSKI, ROBERT S., Ph.D. Michigan State U., Dept. of Botany, East Lansing. *Prof.* (2, 1954)

BANG, FREDERIK B., M.D. Johns Hopkins U., Sch. of Hygiene, Baltimore 5, Md. *Prof. of Pathobiol.; Assoc. Prof. Med.* (4, 1947; 6, 1953)

BARBORIAK, JOSEPH J., Ph.D. Yale U., Nutrition Lab., New Haven 11, Conn. *Research Asst. in Biochem.* (5, 1958)

BARCLAY, RALPH K., Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc.* (2, 1955)

BARD, PHILIP, Ph.D. Johns Hopkins U. Sch. of Med , Dept. Physiol., Baltimore 5, Md. *Prof. and Dir.* (1, 1929)

BARDAWIL, WADI A., M.D. Tufts U. Sch. of Med., Boston 11, Mass. *Asst. Prof. of Pathol., Obstetrics and Gynecol.* (4, 1958)

BARGER, A. CLIFFORD, M.D. Harvard Med. Sch., Dept. of Physiol., 25 Shattuck St., Boston 15, Mass. *Assoc. Prof.* (1, 1950)

BARKER, H. A., Ph.D. U. of California, 337 Biochem.-Virus Lab., Berkeley 4. *Prof. of Microbial Biochem.* (2, 1946)

BARKER, HAROLD G., M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Clin. Surgery.* (1, 1954)

BARKER, S. B., Ph.D. U. of Alabama Med. Ctr., Birmingham 3. *Prof. of Pharmacol.* (1, 1938; 3, 1954)

BARLOW, GEORGE, Ph.D. U. of Tennessee Coll. of Med., Inst. Clin. Invest., Memphis 3. *Asst. Dir., Clin. Physiol. Labs.; Asst. Prof.* (1, 1958)

BARLOW, O. W., M.D., Ph.D. R.F.D. 3, Warwick Rd., Winchester, N.H. (3, 1944)

BARNES, B. O., M.D., Ph.D. 1160 S. Colorado Blvd., Denver 22, Colo. (1, 1932)

BARNES, FREDERICK W., JR., M.D., Ph.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. of Med. and of Physiol. Chem.* (2, 1953)

BARNES, LAVERNE A., Ph.D. Office of Naval Research, Microbiol. Br., Navy Dept., Washington 25, D.C. *Capt. MSC, USN.* (6, 1931)

BARNES, RICHARD HENRY, Ph.D. Cornell U. Grad. Sch. of Nutrition, Ithaca, N.Y. *Prof. and Dean.* (2, 1941; 5, 1944)

BARNES, THOMAS C., D.Sc. Hahnemann Med. Coll. and Hosp., Philadelphia 2, Pa. *Assoc. Prof., Electroencephalographer.* (1, 1942; 3, 1948)

BARNETT, HENRY L., M.D. Albert Einstein Coll. of Med., Dept. of Pediatrics, New York City 61. *Prof. and Chmn. of Dept.* (1, 1952)

BARNUM, CYRUS P., JR., Ph.D. U. of Minnesota, 212 Millard Hall, Minneapolis 14. *Prof. of Physiol. Chem.* (2, 1946)

BAROTT, HERBERT G., E.E. 1307 Noyes Dr., Silver Spring, Md. *Biophysicist.* (5R, 1938)

BARRETT, HAROLD W., Ph.D. U. of Kansas Sch. of Med., Dept. of Biochem., Lawrence. *Assoc. Prof.* (2, 1956)

BARRON, DONALD H., Ph.D. Yale U. Sch. of Med., New Haven, Conn. *Prof. of Physiol.* (1, 1943)

BARRY, GUY T., Ph.D. U. of Tennessee Mem. Research Ctr., Knoxville. *Research Prof.* (2, 1957)

BARTLETT, GRANT R., Ph.D. Scripps Clinic and Research Fndn., La Jolla, Calif. *Research Assoc.* (2, 1951)

BARTLETT, PAUL D., Ph.D. Edsel B. Ford Inst. for Med. Research, Detroit 2, Mich. *Head, Bioorganic Div., Biochem. Dept.* (2, 1950)

BARTLETT, ROSCOE G., JR., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Physiologist.* (1, 1956)

BARTLEY, S. HOWARD, Ph.D. 348 Cowley, East Lansing, Mich. *Prof. of Psychol., Michigan State U.* (1, 1935)

BASINSKI, DANIEL H., Ph.D. Henry Ford Hosp., Dept. of Labs., Detroit 2, Mich. *Assoc. Chemist.* (2, 1953)

BASS, ALLAN D., M.D. Vanderbilt U. Sch. of Med., Dept. of Pharmacol., Nashville, Tenn. *Prof. & Head of Dept.* (3, 1944)

BASS, DAVID E., Ph.D. QM Research & Engin. Ctr., Natick, Mass. *Chief, Physiol. Br.; Assoc. Prof. Physiol., Boston U.* (1, 1955)

BASS, WILLIAM B., Ph.D. The Upjohn Co., Biol. Screening Office, Kalamazoo, Mich. *Research Pharmacologist.* (3, 1955)

BATCHELDER, ESTHER L., Ph.D. 8 Devon Rd., Silver Spring, Md. *Dir., Clothing and Housing Research Div., USDA.* (5, 1933)

BATEMAN, JOHN B., Ph.D. Fort Detrick, Frederick, Md. *Chief, Biophysics Br.* (1, 1945)

BATES, ROBERT W., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Hormone Chemist.* (2, 1936)

BATSON, HERBERT C., Ph.D. 1800 W. Fillmore St., Chicago 12. *Prof. of Biostatistics, U. of Ill. Coll. of Med.* (6, 1949)

BATTERMAN, ROBERT C., M.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York. *Assoc. Prof., Med., Pharmacol & Physiol.* (3, 1941)

BATTERSBY, WILLIAM STANLEY, Ph.D. Mt. Sinai Hosp., Dept. of Neurol., New York City 29. *Research Assoc. in Neurol.* (1, 1957)

BAUER, J. H., M.D. The Rockefeller Fndn., 49 W. 49th St., New York City 20. (4, 1935)

BAUER, ROBERT O., M.D. U. of California Med. Ctr., Dept. Surgery & Anesthesia, Los Angeles 24. (3, 1955)

BAUER, WALTER, M.D. Massachusetts Gen. Hosp., Boston 14. *Chief, Med. Serv.; Jackson Prof. Clin. Med., Harvard Med. Sch.* (1, 1929)

BAUERNFEIND, J. C., Ph.D. Hoffman-LaRoche, Inc., Nutley 10, N. J. *Chief of Applied Nutrition.* (5, 1947)

BAUMANN, CARL A., Ph.D. U. of Wisconsin, Biochem. Dept., Madison 6. *Prof.* (2, 1938; 5, 1938)

BAUMANN, EMIL J., Ph.D. 7 Church Lane, Scarsdale, N.Y. (2R, 1922)

BAUMBERGER, J. PERCY, Sc.D. Stanford U., Physiol. Dept., Stanford, Calif. *Prof., Emer.* (1R, 1921)

BAVETTA, LUCIEN A., Ph.D. U. of Southern California Sch. of Dent., Los Angeles. *Prof. of Biochem. and Nutrition.* (5, 1956)

BAXTER, JAMES H., M.D. Natl. Heart Inst., Lab. of Metab., NIH, Bethesda 14, Md. (3, 1948)

BAYNE-JONES, STANHOPE, M.D. 2330 Massachusetts Ave., N.W., Washington 8, D.C. (4R, 1927; 6R, 1917)

BEACH, ELIOT F., Ph.D. Metropolitan Life Insurance Co., 1 Madison Ave., New York City 10. *Dir., Biochem. Lab.* (2, 1941; 5, 1942)

BEADLE, BUELL W., Ph.D. Midwest Research Inst., 425 Volker Blvd., Kansas City 10, Mo. *Mgr., Chem. Div.* (2, 1947)

BEAMER, PARKER R., Ph.D., M.D. Indiana U. Sch. of Med., Indianapolis. *Prof. of Pathol.* (4, 1955)

BEAN, JOHN W., Ph.D., M.D. U. of Michigan, Ann Arbor. *Prof. of Physiol.* (1, 1932)

BEARD, HOWARD H., Ph.D. 5520 Locke Ave., Fort Worth, Tex. (2, 1928)

BEARD, JOSEPH W., M.D. Duke Hosp., Durham, N. C. *Prof. of Surgery; Assoc. Prof. of Virol.* (4, 1938; 6, 1940)

BEATON, JOHN R., Ph.D. Defence Research Bd., Med. Sec., Ottawa, Canada. *Sci. Staff Officer.* (2, 1955)

BEATTY, CLARISSA H., Ph.D. U. of Oregon Med. Sch., Portland 1. *Asst. Prof.* (1, 1949)

BECK, CLAUDE S., M.D. Lakeside Hosp., Cleveland, Ohio. *Assoc. Surgeon; Prof. of Cardiovasc. Surgery, Western Reserve U.* (4, 1930)

BECK, IVAN THOMAS, C.E.M., M.D., D.I.M. St. Mary's and Royal Victoria Hosps., Dept. of Med., Montreal, Canada. (3, 1951)

BECK, LYLE V., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Physiol. & Pharmacol., Pittsburgh 13, Pa. *Assoc. Prof.* (1, 1941; 3, 1956)

BECK, WILLIAM S., M.D. Massachusetts Gen. Hosp., Boston 14. *Asst. Prof. Med., Tutor in Biochem. Sci., Harvard U.* (2, 1957)

BECKER, D. E., Ph.D. U. of Illinois, Dept. of Animal Sci., Urbana. *Assoc. Prof.* (5, 1956)

BECKER, E. LOVELL, M.D. New York Hosp.-Cornell Med. Ctr., New York 21. *Asst. Prof. of Med.* (1, 1956)

BECKER, ELMER L., Ph.D., M.D. Walter Reed Army Inst. of Research, Washington 12, D.C. *Chief, Dept. of Immunochem.* (6, 1952)

BECKER, R. FREDERICK, Ph.D. Duke U. Sch. of Med., Dept. of Anatomy, Durham, N. C. *Assoc. Prof.* (1, 1949)

BECKER, ROBERT R., Ph.D. Columbia U., New York City 27. *Asst. Prof. of Chem.* (2, 1957)

BECKFIELD, WILLIAM J., M.D. U. of Pennsylvania Grad. Sch. of Med., Philadelphia 4. *Asst. Prof. of Pathol.* (4, 1957)

BECKMAN, EDWARD LOUIS, M.D. Inst. of Aviation Med., Royal Aircraft Estab., Farnborough, Hants, England. *Med. Officer.* (1, 1955)

BECKMAN, HARRY, M.D. Marquette U. Sch. of Med., Milwaukee 3, Wis. *Dir., Dept. of Pharmacol.* (3, 1937)

BEECHER, HENRY K., M.D. Massachusetts Gen. Hosp., Boston 14. *Anesthetist-in-Chief; Dorr Prof. of Research, Harvard U.* (1, 1951; 3, 1940)

BEERS, ROLAND F., JR., M.D., Ph.D. Children's Hosp., Inc., Robert W. Johnson Lab., Baltimore 11, Md. *Dir. of Research.* (2, 1957)

BEERSTECHER, ERNEST, JR., Ph.D. U. of Texas Sch. of Dent., P. O. Box 20068, Houston 25. *Prof. of Biochem.* (2, 1950)

BEESON, W. M., Ph.D. Purdue U., Dept. of Animal Sci., Lafayette, Ind. *Prof.* (5, 1952)

BEEVERS, HARRY, Ph.D. Purdue U., Dept. of Biol. Sci., Lafayette, Ind. *Prof. of Plant Physiol.* (2, 1956)

BEGG, R. W., M.D., D. Phil. U. of Saskatchewan, Dept. of Cancer Research, Saskatoon, Canada. *Prof.* (2, 1954)

BEHER, WILLIAM T., Ph.D. Henry Ford Hosp., Detroit 2, Mich. *Research Assoc., Edsel B. Ford Inst. for Med. Research and Wayne U.* (2, 1955)

BEHNKE, ALBERT R., M.D. U. S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Radiol. Med. Dir.* (1, 1946)

BEHRE, JEANETTE ALLEN, Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Asst. Prof.* (2, 1925)

BEHRENS, OTTO K., Ph.D. Lilly Research Labs., Eli Lilly & Co., Indianapolis 6, Ind. *Dir., Biochem. Research Div.* (2, 1949)

BEIDLER, LLOYD M., Ph.D. Florida State U., Div. of Physiol., Tallahassee. *Prof.* (1, 1955)

BEINERT, HELMUT, Dr. rer. nat. U. of Wisconsin, Inst. for Enzyme Research, 1702 University Ave., Madison 5. *Asst. Prof.* (2, 1953)

BEISER, SAM M., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Microbiol.* (6, 1953)

BELDING, DAVID L., M.D. Boston U. Sch. of Med., Boston, Mass. *Prof. Emer. of Bacteriol. and Exptl. Pathol.* (4, 1927)

BELDING, HARWOOD S., Ph.D. U. of Pittsburgh, Grad. Sch. of Public Health, Pittsburgh 13, Pa. *Prof. of Environ. Physiol.* (1, 1945)

BELFORD, JULIUS, Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Prof.* (3, 1954)

BELKIN, MORRIS, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Cellular Pharmacol. Sec.* (3, 1949)

BELL, E. T., M.D. U. of Minnesota, 101 Jackson Hall, Minneapolis 14. *Prof. Emer. of Pathol.* (4, 1931)

BELL, PAUL HADLEY, Ph.D. American Cyanamid Co., Research Div., Pearl River, N. Y. *Head, Biochem. Dept.* (2, 1957)

BELLVILLE, J. WELDON, M.D. Mem. Ctr. for Cancer & Allied Diseases, New York City 21. *Asst. Clinician.* (3, 1958)

BENACERRAF, BARUJ, M.D. New York U. Coll. of Med., New York City 16. *Asst. Prof. of Pathol.* (6, 1957)

BENCOSME, SERGIO A., M.D. U. of California Sch. of Med., Dept. of Pathol., Los Angeles 24. (4, 1956)

BENDER, M. B., M.D. New York U. Coll. of Med., New York City. *Prof. of Clin. Neurol.; Dir., Neurol., Mt. Sinai Hosp.* (1, 1947)

BENDICH, A., Ph.D. Sloan-Kettering Inst., New York 21. *Head, Sec. Organ. Biochem.; Assoc. Prof. Biochem., Sloan-Kettering Div., Cornell U.* (2, 1950)

BENDITT, EARL P., M.D. U. of Washington, Seattle 5. *Prof. of Pathol.* (4, 1947)

BENEDICT, ALBERT A., Ph.D. U. of Kansas, Dept. of Bacteriol., Lawrence. *Assoc. Prof., Immunol. and Immuno-chem.* (6, 1958)

BENENSON, ABRAM S., M.D. Walter Reed Army Inst. of Research, Div. of Immunol., Washington 12, D. C. *Dir.* (6, 1956)

BENESCH, REINHOLD, Ph.D. Marine Biol. Lab., Woods Hole, Mass. (2, 1952)

BENESCH, RUTH E., Ph.D. Marine Biol. Lab., Woods Hole, Mass. (2, 1957)

BENFEY, BRUNO GEORGE, M.D. McGill U., Dept. of Pharmacol., Montreal, Canada. (3, 1958)

BENFORADO, JOSEPH M., M.D. U. of Buffalo, Dept. of Pharmacol., Buffalo, N. Y. *Assoc. Prof.* (3, 1957)

BENHAM, OLIVE RAY, B.S. Connecticut State Dept. of Health, Bur. of Labs., Hartford. *Chief Serologist.* (6, 1944)

BENIRSCHKE, KURT, M.D. Boston Lying-In Hosp., Boston 15, Mass. *Pathologist; Assoc. in Pathol., Harvard Med. Sch.* (4, 1956)

BENJAMIN, FRED B., D.M.D., Ph.D. U. of Pennsylvania, Sch. of Med., Philadelphia 4. *Asst. Prof. of Physiol.* (1, 1955)

BENNETT, A. LAWRENCE, Ph.D., M.D. U. of Nebraska Coll. of Med., Dept. of Physiol. & Pharmacol., Omaha. *Prof. and Asst. Chmn.* (1, 1941)

BENNETT, EDWARD L., Ph.D. U. of California Radiation Lab., Berkeley 4. *Research Chemist.* (2, 1955)

BENNETT, GRANVILLE A., M.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Pathol. and Dean, Coll. of Med.* (4, 1931)

BENNETT, H. STANLEY, M.D. U. of Washington Sch. of Med., Seattle 5. *Prof. of Anatomy.* (1, 1946)

BENNETT, IVAN L., JR., M.D. Johns Hopkins Hosp., 1140 RUI Bldg., Baltimore 5, Md. *Head, Biol. Div., Dept. of Med.* (6, 1954)

BENNETT, LESLIE L., M.D., Ph.D. U. of California Med. Ctr., San Francisco 22. *Prof. of Physiol.* (1, 1945)

BENNETT, LESLIE R., M.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Radiol.* (4, 1956)

BENNETT, MARY ADELIA, Ph.D. Inst. for Cancer Research, Fox Chase, Philadelphia 11, Pa. *Research Biochemist.* (2, 1941)

BENNETT, WARREN A., M.D. Thomas D. Dee Hosp., Ogden, Utah. *Dir. of Labs. and Chief of Pathol.* (4, 1952)

BENSON, ANDREW A., Ph.D. Pennsylvania State U., Dept. of Agric. and Biol. Chem., University Park. *Assoc. Prof.* (2, 1955)

BENSON, CLARA C., Ph.D. 160 Dorset St., West, Port Hope, Ontario, Canada. *Prof. Emer. of Food Chem., U. of Toronto.* (2R, 1906)

BENSON, OTIS O., JR., M.D. USAF Sch. of Aviation Med., Randolph AFB, Tex. *Maj. Gen. USAF (MC); Commandant.* (1, 1951)

BENSON, WILBUR M., Ph.D., M.D. U. of Minnesota Med. Sch., Dept. of Pharmacol., Minneapolis 14. *Prof.* (3, 1954)

BENTLEY, ORVILLE G., Ph.D. Ohio Agric. Expt. Sta., Dept. of Animal Sci., Wooster. *Prof.* (5, 1958)

BENTLEY, RONALD, Ph.D. U. of Pittsburgh Grad. Sch. of Public Health, Pittsburgh 13, Pa. *Assoc. Prof. of Biochem.* (2, 1954)

BENTON, JOSEPH G., Ph.D., M.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof., Physical Med. & Rehabilitation.* (3, 1949)

BENZINGER, THEODOR, M.D., D.Sc. Naval Med. Research Inst., Bethesda 14, Md. *Head, Bio-Energetics Div.* (1, 1950)

BERG, BENJAMIN N., M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. in Patbol.* (4, 1928)

BERG, CLARENCE P., Ph.D. State U. of Iowa, Dept. of Biochem., Iowa City. *Prof.* (2, 1933; 5, 1936)

BERG, PAUL, Ph.D. Washington U. Sch. of Med., Dept. of Microbiol., St. Louis 10, Mo. *Assoc. Prof.* (2, 1955)

BERGE, TRYGVE O., Ph.D. USA Med. Unit, Fort Detrick, Md. *Chief, Microbiol. Br.* (6, 1954)

BERGEIM, OLAF, Ph.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Biochem.* (1R, 1916; 2R, 1914)

BERGEN, JOHN R., Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Staff Member.* (1, 1955)

BERGER, EUGENE Y., M.D. 3640 Johnson Ave., New York City 63. *Asst. Prof. of Med., N. Y. U. Coll. of Med.* (1, 1953)

BERGER, F. M., M.D. 227 Prospect Ave., Princeton, N. J. *Dir., Wallace Labs., New Brunswick, N. J.* (3, 1949)

BERGERON, GEORGES A., M.D. Laval U. Med. Sch., Dept. of Physiol., Quebec, Canada. *Prof.* (1, 1955)

BERGMANN, WERNER, Ph.D. Yale U., Sterling Chem. Lab., New Haven, Conn. *Prof. of Chem.* (2, 1934)

BERLIN, NATHANIEL I., M.D., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Metab. Serv.* (1, 1954)

BERLINER, ROBERT W., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Assoc. Dir. in charge of Research.* (1, 1950)

BERMAN, DAVID T., Ph.D. U. of Wisconsin, Veterinary Sci. Dept., Madison 6. *Prof.* (6, 1955)

BERNARD, RICHARD, Ph.D. Laval U., Dept. of Biol., Blvd. de l'Entente, Quebec, Canada. *Prof. of Animal Physiol.* (1, 1947)

BERNE, ROBERT M., M.D. Western Reserve U. Sch. of Med., Dept. of Physiol., Cleveland 6, Ohio. *Assoc. Prof.* (1, 1951)

BERNFELD, PETER, Ph.D. Bio-Research Inst., Cambridge 41, Mass. *Dir. of Research.* (2, 1952)

BERNHART, FINN W., Ph.D. Wyeth Inst. for Med. Research, Nutrition Dept., Radnor, Pa. *Head.* (5, 1958)

BERNHEIM, FREDERICK, Ph.D. Duke U. Sch. of Med., Box 3109, Durham, N.C. *Prof. of Pharmacol.* (2, 1933; 3, 1935)

BERNHEIMER, ALAN WEYL, Ph.D. New York U. Coll. of Med., New York City 16. *Prof. of Microbiol.* (6, 1951)

BERNSTEIN, I. A., Ph.D. University Hosp., R6649 Kresge, Ann Arbor, Mich. *Asst. Prof. Biol. Chem., U. of Michigan.* (2, 1956)

BERNTHAL, THEODORE G., M.D. Med. Coll. of South Carolina, Dept. of Physiol., Charleston 16. *Prof. and Head of Dept.* (1, 1932)

BERRY, GEORGE PACKER, M.D. Harvard Med. Sch., Boston 15, Mass. *Dean and Prof. of Bacteriol.* (6, 1934)

BERRYMAN, GEORGE H., Ph.D., M.D. Abbott Labs., No. Chicago, Ill. *Dir., Med. Sci.; Clin. Asst. Prof. of Med., U. of Illinois.* (1, 1949)

BERSON, SOLOMON A., M.D. VA Hosp., New York City 68. *Chief, Radioisotope Serv.* (1, 1957)

BESSEY, OTTO A., Ph.D. QM Research & Engin. Ctr., Natick, Mass. *Assoc. Chief for Research.* (2, 1938; 5, 1943)

BESSMAN, SAMUEL P., M.D. University Hosp., Baltimore 1, Md. *Assoc. Prof. of Pediatrics, U. of Maryland.* (2, 1954)

BEST, CHARLES H., M.D., D.Sc. U. of Toronto, Toronto, Canada. *Dir., Banting & Best Dept. of Med. Res. & Dept. of Physiol.* (1, 1923; 2, 1923)

BETHELL, FRANK H., M.D. 409 Lenawee Dr., Ann Arbor, Mich. *Prof. Int. Med.; Dir., Thomas Henry Simpson Mem. Inst.* (4, 1936)

BETHKE, ROLAND M., Ph.D. Ralston Purina Co., 835 S. 8th St., St. Louis 2, Mo. *Vice Pres. and Dir. of Research.* (2, 1928; 5, 1933)

BEUTNER, R. H., Ph.D., M.D. Des Moines Still Coll., Med. Res. Lab., Des Moines, Iowa. *Dir.; Assoc. Prof., Pharmacol.* (1R, 1924; 3, 1924)

BEVANS, MARGARET, New York Infirmary, 15th St. & Stuyvesant Sq., E., New York City. (4, 1952)

BEVERIDGE, J. M. R., Ph.D., M.D. Queen's U., Kingston, Canada. *Craine Prof. and Head, Dept. of Biochem.* (5, 1957)

BEYER, KARL H., JR., Ph.D., M.D. Merck Sharp & Dohme Research Labs., West Point, Pa. *Vice-Pres. for Life Sci.* (1, 1942; 3, 1944)

BICKFORD, REGINALD G., M.D. Mayo Clinic, Rochester, Minn. *Head, Lab. of Neurophysiol.; Prof. of Physiol., U. of Minn.* (1, 1949)

BIDDULPH, CLYDE, Ph.D. Utah State U., Logan. *Prof. of Physiol.* (1, 1952)

BIER, MILAN, Ph.D. 2421 Webb Ave., New York City 68. *Asst. Prof., Fordham U.* (2, 1956)

BIER, OTTO, M.D. Escola Paulista de Medicina, Caixa Postal 7144, São Paulo, Brazil. *Head.* (6, 1947)

BIERI, JOHN G., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, Lab. of Nutrition & Endocrinol., NIH, Bethesda 14, Md. *Biochemist.* (5, 1954)

BIERMAN, HOWARD R., M.D. City of Hope Med. Ctr., Duarte, Calif. *Med. and Sci. Dir.* (3, 1950)

BIETER, RAYMOND N., M.D., Ph.D. U. of Minnesota, Dept. of Pharmacol., Minneapolis 14. *Prof. and Head of Dept.* (3, 1930)

BILLS, CHARLES E., Ph.D. 6403 Murray Hill Rd., Baltimore 12, Md. *Consultant.* (2, 1928; 5, 1935)

BING, FRANKLIN C., Ph.D. 30 N. La Salle St., Chicago 2, Ill. *Consulting Chemist.* (2, 1931; 5, 1934)

BING, RICHARD J., M.D. Washington U. Sch. of Med., St. Louis, Mo. *Prof. of Med.* (1, 1942)

BINKLEY, FRANCIS, Ph.D. Emory U., Dept. of Biochem., Atlanta 22, Ga. *Prof.* (2, 1947)

BINKLEY, STEPHEN BENNETT, Ph.D. U. of Illinois Coll. of Med., Chicago 11. *Prof. of Biochem.* (2, 1941)

BIRD, FRANCIS H., Ph.D. Eastern States Farmers' Exch., Inc., Feed Research Ctr., Ellington, RFD 1, Conn. *Feed Research.* (5, 1951)

BIRD, HERBERT R., Ph.D. U. of Wisconsin Coll. of Agric., Dept. of Poultry Husbandry, Madison 6. *Prof.* (5, 1947)

BIRD, ORSON D., Ph.D. Parke, Davis & Co., Research Labs., Detroit 32, Mich. *Lab. Dir. in Nutritional Research.* (2, 1947)

BIRD, ROBERT M., M.D. University Hosp., Dept. of Med., Oklahoma City, Okla. *Assoc. Prof. of Med. and of Physiol.* (1, 1950)

BIRNBAUM, SANFORD M., Ph.D. Natl. Cancer Inst., Lab. of Biochem., NIH, Bethesda 14, Md. *Sr. Scientist.* (2, 1953)

BIRNIE, JAMES H., Ph.D. Morehouse Coll., Dept. of Biol., Atlanta, Ga. *Prof. and Chmn. of Dept.* (1, 1952)

BIRREN, JAMES E., Ph.D. Natl. Inst. of Mental Health, NIH, Bethesda 14, Md. *Chief, Sec. on Aging.* (1, 1950)

BISBEY, BERTHA, Ph.D. 1402 Bass Ave., Columbia, Mo. *Prof. of Nutrition, U. of Missouri.* (5R, 1933)

BISCHOFF, FRITZ E., Ph.D. Santa Barbara Cottage Hosp., Santa Barbara, Calif. *Dir. of Research.* (2, 1928; 5, 1933)

BISHOP, GEORGE H., Ph.D. Washington U. Med. Sch., St. Louis 10, Mo. *Prof. Emer. of Neurophysiol.* (1, 1923)

BISHOP, JACK G., Ph.D. Baylor U. Coll. of Dent., Dept. of Physiol., Dallas 10, Tex. *Prof.; Prof. and Chmn., Grad. Res. Inst.* (1, 1957)

BISKIND, G. R., M.D. 450 Sutter St., San Francisco, Calif. *Chief Pathol., Mt. Zion Hosp.; Assoc.*

Prof. Pathol., U. Calif. (4, 1944)

BLACK, ALEX, Ph.D. Pennsylvania State U., Agric. Expt. Sta., University Park. *Asst. Dir.* (5, 1947)

BLACK, ARTHUR L., Ph.D. U. of California Sch. of Veterinary Med., Davis. *Assoc. Prof., Biochem.; Assoc. Biochemist, Expt. Sta.* (2, 1956)

BLACK, EDGAR C., Ph.D. U. of British Columbia, Dept. of Physiol., Vancouver, Canada. *Assoc. Prof. of Physiol.* (1, 1943)

BLACK, FRANCIS L., Ph.D. Yale U. Sch. of Med., New Haven, Conn. *Research Assoc. in Epidemiol.; Lect. in Microbiol.* (6, 1957)

BLACK, SIMON, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Sec. on Biochem.* (2, 1948)

BLACKMORE, WILLIAM P., Ph.D. U. of Texas, Southwestern Med. Sch., Dept. of Pharmacol., Dallas 19. *Assoc. Prof.* (3, 1957)

BLAIR, EDGAR A., Ph.D. 789 Burr Rd., San Antonio 9, Tex. (1, 1936)

BLAIR, HENRY A., Ph.D. U. of Rochester Sch. of Med. & Dent., Rochester 20, N.Y. *Dir., Dept. Radiation Biol.; Dir., Atomic Energy Proj.* (1, 1934)

BLAIR, JOSEPH REESE, M.D. Army Med. Research Lab., Ft. Knox, Ky. *Lt. Col.* (1, 1952)

BLAIR, MURRAY R., JR., Ph.D. Buffalo U. Med. Sch., Dept. of Pharmacol., Buffalo, N.Y. *Asst. Prof.* (3, 1957)

BLAKE, WILLIAM D., M.D. U. of Oregon Med. Sch., Dept. of Physiol., Portland 1. *Assoc. Prof. of Physiol.* (1, 1950)

BLAKEMORE, WILLIAM STEPHEN, M.D. U. of Pennsylvania Sch. of Med. Philadelphia 4. *Asst. Prof. of Surgery.* (1, 1957)

BLANCHARD, KENNETH C., Ph.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Pharmacol.* (3, 1954)

BLANKENHORN, DAVID H., M.D. Los Angeles County Gen. Hosp., Los Angeles, Calif. *Asst. Prof. Int. Med., U. of Southern Calif.* (4, 1957)

BLATHERWICK, NORMAN R., Ph.D. P.O. Box 5415, Daytona Beach, Fla. (1R, 1915; 2R, 1915; 5R, 1934)

BLAU, NATHAN F., Ph.D. VA Ctr., 5500 E. Kellogg, Wichita 8, Kans. *Research Biochemist.* (2, 1928)

BLISH, MORRIS J., Ph.D. 4810 N. 31st St., Phoenix, Ariz. (2R, 1944)

BLISS, CHESTER ITTNER, Ph.D. Connecticut Agric. Expt. Sta., P.O. Box 1106, New Haven. *Biometrician; Lect., Yale U.* (3, 1944)

BLISS, ELEANOR A., Sc.D. Bryn Mawr Coll., Dept. of Biol., Bryn Mawr, Pa. *Prof.; Dean of Grad. Sch.* (6, 1931)

BLIVAISS, BEN B., Ph.D. Chicago Med. Sch., Chicago 12, Ill. *Asst. Prof. of Physiol. and Pharmacol.* (1, 1955)

BLOCH, EDWARD H., M.D., Ph.D. Western Reserve U. Sch. of Med., Dept. of Anatomy, Cleveland 6, Ohio. *Assoc. Prof.* (6, 1956)

BLOCH, HUBERT, M.D. U. of Pittsburgh Med. Sch., Dept. of Microbiol., Pittsburgh 13, Pa. *Prof. and Chmn.* (6, 1954)

BLOCH, KONRAD, Ph.D. Harvard U., Dept. of Chem., Cambridge 38, Mass. *Prof. of Biochem.* (2, 1944)

BLOCK, MATTHEW H., Ph.D., M.D. U. of Colorado Med. Ctr., Denver. *Assoc. Prof. of Med.* (4, 1953)

BLOCK, RICHARD J., Ph.D. 15 Cooper Rd., Scarsdale, N.Y. *Lect. Biochem., New York Med. Coll.; Consultant, Boyce-Thompson Inst.* (2, 1934; 5, 1933)

BLOCK, WALTER D., Ph.D. U. of Michigan Med. Sch., Kresge Med. Research Bldg., Ann Arbor. *Assoc. Prof. of Biol. Chem.* (2, 1942)

BLOOD, FRANK R., Ph.D. Vanderbilt U., Dept. of Biochem., Nashville 4, Tenn. *Prof.* (1, 1950)

BLOODWORTH, J.M.B., JR., M.D. Ohio State U. Coll. of Med., Dept. of Pathol., Columbus 10. *Assoc. Prof.* (4, 1956)

BLOOM, BEN, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Biochemist.* (2, 1955)

BLOOM, WILLIAM, M.D. 5454 So. Shore Dr., Chicago Ill. *Prof. of Anatomy, U. of Chicago.* (4, 1930)

BLOOMFIELD, A. L., M.D. Stanford U. Hosp., San Francisco 15, Calif. *Prof. Emer. of Med.* (3, 1927; 4, 1927)

BLOOR, W. R., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. Emer. of Biochem.* (1R, 1915; 2R, 1910)

BLUM, JACOB J., Ph.D. U. of Michigan, Mental Health Research Inst. and Dept. of Biochem., Ann Arbor. (1, 1955)

BLUMBERG, HAROLD, Sc.D. Endo Labs., Inc., 84-40 101st St., Richmond Hill 18, N.Y. *Dir., Biol. Labs.* (3, 1954; 5, 1942)

BLUMENSTOCK, JULIUS, M.D. VA Hosp., 1900 E. Main St., Danville, Ill. (1, 1925)

BLUMENTHAL, HERMAN T., Ph.D., M.D. The Jewish Hosp., 216 S. Kingshighway, St. Louis 10, Mo. *Dir., Inst. of Exptl. Pathol.* (4, 1951)

BLUNT, J. WALLACE, JR., M.D. St. Luke's Hosp., Cleveland 4, Ohio. *Dir. of Surgical Research.* (4, 1957)

BLY, CHAUNCEY G., M.D., Ph.D. Highland Hosp., Rochester 20, N.Y. *Pathologist; Assoc. Clin. Prof. of Pathol., U. of Rochester.* (4, 1951)

BOBB, J. RICHARD R., M.D. Bowman Gray Sch. of Med., Wake Forest Coll., Winston-Salem, N.C. *Assoc. Prof., Physiol. and Pharmacol.* (1, 1949)

BOCK, JOSEPH C., Ph.D. 2324 N. 46th St., Milwaukee 10, Wis. *Prof. Emer. Biochem., Marquette U.; Biochemist, County Hosp.* (2R, 1916)

BOCK, ROBERT M., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Assoc. Prof.* (2, 1957)

BODANSKY, AARON, Ph.D. 7002-58th Ave., N.E., Seattle 15, Wash. (2R, 1926)

BODANSKY, OSCAR, Ph.D., M.D. Sloan-Kettering Inst., New York 21. *Chief, Div. Metab.; Chief, Dept. Biochem., Mem. Hosp.* (2, 1937; 3, 1942)

BODIAN, DAVID, Ph.D., M.D. Johns Hopkins U., Baltimore 5, Md. *Prof. of Anatomy and Dir. of Dept.* (6, 1949)

BOELL, EDGAR J., Ph.D. Yale U., Gibbs Lab., New Haven, Conn. *Ross G. Harrison Prof. of Exptl. Zool.* (1, 1942)

BOETTIGER, EDWARD G., Ph.D. U. of Connecticut, Box U-42, Storrs. *Assoc. Prof. of Zool.* (1, 1950)

BOGDANSKI, DONALD F., Ph.D. 4506 Harrison St., N.W., Washington 15, D.C. *Sr. Asst. Scientist, Natl. Heart Inst., NIH.* (3, 1957)

BOGER, WILLIAM P., M.D. Norristown State Hosp.,

Norristown, Pa. *Dir. of Research.* (3, 1948)

BOGERT, L. JEAN, Ph.D. Hotel Claremont, Berkeley, Calif. (2R, 1917)

BOHR, DAVID F., M.D. U. of Michigan, Physiol. Lab., Ann Arbor. *Prof.* (1, 1949)

BOHR, VERNON CLARENCE, Ph.D. Coll. of Med. Evangelists, Loma Linda, Calif. *Asst. Prof. of Physiol.* (1, 1957)

BOLLIGER, ADOLPH, D.Sc. U. of Sydney, Gordon Craig Research Lab., Sydney, Australia. *Dir. of Research.* (2, 1928)

BOLLMAN, J. L., M.D. Mayo Clinic, Rochester, Minn. *Prof. of Physiol., Mayo Fndn.* (4, 1927)

BOND, GLENN C., Ph.D., M.D. The Upjohn Co., Kalamazoo 99, Mich. *Asst. Dir. of Control.* (6, 1939)

BOND, VICTOR P., M.D. Brookhaven Natl. Lab., Med. Dept., Upton, N. Y. *Head, Div. of Microbiol.; Physician; Radiobiologist.* (1, 1957)

BONDI, AMEDEO, JR., Ph.D. Hahnemann Med. Coll., Dept. of Microbiol., Philadelphia 2, Pa. *Prof. and Head of Dept.* (6, 1948)

BONNER, DAVID M., Ph.D. Yale U., Microbiol. Dept., New Haven 11, Conn. *Prof.* (2, 1948)

BONNER, JAMES, Ph.D. California Inst. of Tech., Pasadena 4. *Prof. of Biol.* (2, 1952)

BONNYCASTLE, DESMOND D., M.D., Ph.D. Seton Hall Coll. of Med. and Dent., Jersey City 4, N. J. *Prof. & Chmn., Pharmacol.* (3, 1947)

BONSNES, ROY W., Ph.D. Cornell U. Med. Coll., Dept. of Biochem., New York City 21. *Assoc. Prof.* (2, 1947)

BONTING, SJOERD L., Ph.D. Presbyterian-St. Luke's Hosp., Chicago 12, Ill. *Asst. Prof., Dept. Biol. Chem., U. of Illinois Coll. of Med.* (2, 1958)

BOOHER, LELA E., Ph.D. 4858 E. 13th St., Tucson, Ariz. *Consultant.* (2, 1933; 5, 1933)

BOOKER, WALTER M., Ph.D. Howard U. Sch. of Med., Dept. of Pharmacol., Washington 2, D. C. *Head.* (1, 1948; 3, 1948)

BOONE, BERT R., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Spec. Projects Officer.* (1, 1951)

BOONE, IRENE J. U., M.D. U. of California, Los Alamos Sci. Lab., Los Alamos, N. Mex. *Staff Member, Biomed. Res. Group.* (2, 1957)

BOOR, ALDEN K., Ph.D. 609½ Cherry St., Lansing, Mich. (2R, 1931)

BOOTH, ALBERT N., Ph.D. USDA, Western Utilization Res. and Develop. Div., Albany 10, Calif. *Acting Head, Biochem. Investigations.* (2, 1958)

BORCHERS, RAYMOND, Ph.D. U. of Nebraska, Dept. of Biochem. and Nutrition, Lincoln 3. *Assoc. Prof.* (5, 1951)

BORDLEY, JAMES, III, M.D. Mary Imogene Bassett Hosp., Cooperstown, N. Y. *Dir.; Clin. Prof. of Med., Columbia U.* (1, 1938)

BOREK, ERNEST, Ph.D. City Coll. of New York, Dept. of Chem., Convent Ave. and 140th St., New York City. *Assoc. Prof.* (2, 1947)

BORISON, HERBERT LEON, Ph.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City 12. *Assoc. Prof.* (1, 1950; 3, 1952)

BOROFF, DANIEL A., Ph.D. New England Inst. for Med. Research, Ridgefield, Conn. (6, 1947)

BORSOOK, HENRY, M.D., Ph.D. California Inst. of Tech., Pasadena 4. *Prof. of Biochem.* (2, 1931)

BOSS, WILLIS R., Ph.D. Syracuse U., Dept. of Zool., Syracuse, N. Y. *Prof.* (1, 1954)

BOSSHARDT, DAVID K., Ph.D. Merck Inst. for Therap. Research, Rahway, N. J. *Research Assoc. in Nutrition.* (5, 1947)

BOSTICK, WARREN L., M.D. U. of California Med. Sch., Dept. of Pathol., San Francisco 22. *Assoc. Prof.; Dir., Clin. Labs.* (4, 1949)

BOSWORTH, ALFRED WILLSON, A.M. R.F.D. 4, Circleville, Ohio. *Consulting Chemist.* (2R, 1936; 5R, 1935)

BOTELHO, STELLA Y., M.D., U. of Pennsylvania, Grad. Sch. of Med., Dept. of Physiol. and Pharmacol., Philadelphia 4. *Assoc. Prof.* (1, 1954)

BOTT, PHYLLIS A., Ph.D. Woman's Med. Coll., Dept. of Physiol. Chem., Philadelphia 29. *Prof. & Chmn.* (1, 1957; 2, 1938)

BOTTS, DOROTHY JEAN, Ph.D. Dartmouth Med. Sch., Dept. of Biochem., Hanover, N. H. (1, 1952)

BOUCHER, ROBERT V., Ph.D. Pennsylvania State U., 303 Frear Labs., University Park. *Prof. of Agric. and Biol. Chem.* (5, 1945)

BOUMAN, H. D., M.D. U. of Wisconsin Med. Sch., Madison 6. *Prof. of Physical Med.* (1, 1943)

BOURDILLON, JAQUES, M.D. New York State Dept. of Health, New Scotland Ave., Albany 1. *Sr. Med. Biochemist.* (2, 1956)

BOURNE, WESLEY, M.D., M.Sc. McGill U., Montreal, Canada. *Consultant Anesthetist; Emer. Prof. of Anesthesia.* (3, 1936)

BOURQUE, JOSEPH E., M.D., Ph.D. 1240 Edgewood Ave., Chicago Heights, Ill. *Physician and Surgeon, Private Practice.* (1, 1949)

BOURQUIN, HELEN, Ph.D. 1331 N. Tejon St., Colorado Springs, Colo. (1, 1925)

BOVARNICK, MARIANNA R., Ph.D. VA Hosp., Brooklyn 9, N. Y. *Lect., State U. of New York Coll. of Med.* (2, 1953)

BOWEN, WILLIAM J., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Physiologist; Scientist Dir.* (1, 1948)

BOWMAN, DONALD E., Ph.D. Indiana U. Sch. of Med., Dept. of Biochem., Indianapolis 7. *Prof. and Chmn. of Dept.* (2, 1944)

BOWMAN, KATHERINE L., B.A. Jewish Hosp. of Brooklyn, Allergy Lab., Brooklyn 38, N. Y. *Research Assoc. in Allergy.* (6, 1946)

BOXER, GEORGE E., Ph.D. 513 Fairmont Ave., Westfield, N. J. *Dir. of Enzyme Research, Merck Inst. for Therap. Research.* (2, 1946)

BOYARSKY, LOUIS L., Ph.D. U. of Kentucky, Dept. of Anatomy and Physiol., Lexington. *Assoc. Prof.* (1, 1954)

BOYD, ELDON M., M.D. Queen's U., Kingston, Canada. *Prof. and Head, Dept. of Pharmacol.* (3, 1941)

BOYD, EUGENE STANLEY, Ph.D. U. of Rochester Sch. of Med. & Dent., Dept. of Pharmacol., Rochester 20, N. Y. *Asst. Prof.* (3, 1958)

BOYD, LINN J., M.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City 29. *Prof. of Pharmacol.; Dir. of Med.* (3, 1950)

BOYD, M. JOHN, Ph.D. Hahnemann Med. Coll., Philadelphia 2, Pa. *Prof. and Chmn., Dept. of Biol. Chem.* (2, 1947)

BOYD, T. E., Ph.D. 9 Walworth Ave., Scarsdale, N. Y. *Asst. Dir. of Research, Natl. Fndn. for Infantile Paralysis.* (1, 1924)

BOYD, WILLIAM C., Ph.D. Boston U. Sch. of Med., Boston 18, Mass. *Prof.* (6, 1933)

BOYDEN, ALAN A. Rutgers, The State U., New Brunswick, N. J. *Prof. and Chmn. of Zool.* (6, 1948)

BOYDEN, EDWARD A., Ph.D. U. of Washington, Dept. of Anatomy, Seattle 5. *Research Prof.; Emer. Prof., U. of Minnesota.* (1, 1929)

BOYER, PAUL D., Ph.D. U. of Minnesota, Dept. of Physiol. Chem., Minneapolis 14. *Hill Prof. of Enzymol.* (2, 1944)

BOYLE, PAUL E., D.M.D. Western Reserve U. Sch. of Dent. and Med., Cleveland 6, Ohio. *Dean; Prof. of Dent. Med., and Oral Patbol.* (4, 1939)

BOZICEVICH, JOHN, M.A. Natl. Insts. of Health, Bethesda 14, Md. *Sec. Chief, Basic Immunol. Lab.* (6, 1948)

BOZLER, EMIL, Ph.D. Ohio State U., Columbus. *Prof. of Physiol.* (1, 1932)

BRADBURY, JAMES T., Sc.D. University Hosp., Dept. of Obstetrics and Gynecol., Iowa City, Iowa. *Prof.* (1, 1941)

BRADLEY, HAROLD C., Ph.D. 2639 Durant Ave., Berkeley, Calif. (1R, 1911; 2R, 1908)

BRADLEY, STANLEY E., M.D. Columbia U. Coll. of P. & S.. New York City 32. *Prof. of Med.* (1, 1947)

BRADLEY, WILLIAM B., Ph.D. American Inst. of Baking, 400 E. Ontario St., Chicago 11, Ill. *Sci. Research Dir.* (1, 1939)

BRADLOW, H. LEON, Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc.* (2, 1954)

BRADY, ROSCOE O., M.D. Natl. Inst. of Neurological Diseases & Blindness, NIH, Bethesda 14, Md. *Chief, Sec. on Lipid Metab.* (2, 1955)

BRAGDON, JOSEPH HENRY, M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Med. Dir., PHS.* (4, 1950)

BRANCH, E. A. G., M.D. Lancaster Hosp., Lancaster, Canada. *Chief, Lab. Serv.; Provincial Med. Dir., Red Cross Blood Transfusion Serv.* (4, 1929)

BRAND, EUGENE D., M.D. U. of Virginia Med. Sch., Dept. of Pharmacol., Charlottesville. *Asst. Prof.* (3, 1954)

BRANDES, W. W., M.D. Southampton Hosp., Southampton, N. Y. (4, 1931)

BRANHAM, SARA E., Ph.D., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Sec. on Bacterial Toxins, DBS.* (6R, 1926)

BRANION, HUGH DOUGLAS, Ph.D. Ontario Agric. Coll., Guelph, Canada. *Head, Dept. of Nutrition; Chmn., Grad. Studies.* (5, 1938)

BRASSFIELD, CHARLES R., Ph.D. U. of Michigan, Ann Arbor. *Prof. of Physiol.* (1, 1937)

BRATTON, ANDREW CALVIN, JR., M.D., Ph.D. Parke, Davis & Co., Research Labs., Detroit 32, Mich. *Dir. of Pharmacol. Research.* (3, 1941)

BRATZLER, JOHN W., Ph.D. Pennsylvania State U., University Park. *Prof. of Animal Nutrition.* (5, 1958)

BRAUER, RALPH W., Ph.D. U.S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Head, Pharmacol. Br.* (1, 1957; 3, 1948)

BRAUN, HERBERT A., Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D. C. *Pharmacologist.* (3, 1941)

BRAUNSTEIN, HERBERT, M.D. U. of Cincinnati Coll. of Med., Cincinnati, Ohio. *Asst. Prof. of Patbol.* (4, 1957)

BRAUNWALD, EUGENE, M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chief, Sec. of Cardiol., Clinic of Surgery.* (1, 1956)

BRAWERMAN, GEORGE, Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Research Assoc.* (2, 1958)

BRAZDA, FRED G., Ph.D. Louisiana State U. Sch. of Med., New Orleans 12. *Prof. of Biochem.; Head of Dept.* (2, 1954)

BRAZIER, MARY A. B., Ph.D. Mass. Gen. Hosp., Boston 14. *Neurophysiologist; Res. Assoc., Neurol., Harvard; Res. Assoc. Elec. Engin., MIT.* (1, 1947)

BRECHER, GEORGE, M.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Hematol. Serv., Clin. Patbol. Dept.* (4, 1949)

BRECHER, GERHARD A., M.D., Ph.D. Emory U., Div. of Basic Health Sci., Dept. Physiol., Atlanta 22, Ga. *Prof. & Chmn. of Dept.* (1, 1949)

BREEDIS, CHARLES, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Assoc. Prof. of Patbol.* (4, 1948)

BREMER, FREDERIC, M.D. 115 Blvd. de Waterloo, Brussels, Belgium. *Prof. and Dir. of Dept. of Physiopatbol., U. of Brussels.* (1H, 1950)

BRESSANI, RICARDO, Ph.D. Instituto de Nutricion de Centro America y Panama, Guatemala. *Chief, Div. of Agric. and Food Chem.* (5, 1958)

BREWER, CARL R., Ph.D. USA Chem. Corps Research & Development Command, Washington 25, D. C. *Chief, Research Div.* (2, 1948)

BREWER, JOHN H., Ph.D. Hynson Westcott and Dunning, Baltimore, Md. *Dir. of Biol. Research.* (6, 1948)

BREWER, NATHAN R., Ph.D., D.V.M. U. of Chicago, Chicago 37, Ill. *Assoc. Prof. of Physiol.; Supt. of Animal Qtrs.* (1, 1948)

BREWER, WILMA D., Ph.D. Iowa State Coll., Dept. of Foods and Nutrition, Ames. *Prof.* (5, 1951)

BRIDGE, EDWARD M., M.D. U. of Buffalo Sch. of Med., Buffalo 14, N. Y. *Prof. of Pharmacol.* (3, 1951)

BRIDGFORTH, EDWIN B., A.B. Vanderbilt U. Sch. of Med., Dept. of Prev. Med., Nashville, Tenn. *Asst. Prof. of Biostatistics.* (5, 1958)

BRIEGER, HEINRICH, M.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Prof. of Industrial Med.* (3, 1954)

BRIGGS, A. P., M.D. Med. Coll. of Georgia, Augusta. *Prof. Emer. of Biochem.* (2R, 1923)

BRIGGS, DAVID R., Ph.D. U. of Minnesota, Div. of Agric. Biochem., University Farm, St. Paul 1. *Prof.* (2, 1946)

BRIGGS, FRED N., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. in Pharmacol.* (3, 1957)

BRIGGS, GEORGE M., Ph.D. Natl. Insts. of Health, Lab. of Nutrition and Endocrinol., Bethesda 14, Md. *Chief, Nutrition Unit.* (5, 1947)

BRIN, MYRON, Ph.D. Food and Drug Research Labs., Inc., Maspeth 78, New York City. *Chief Biologist.* (2, 1955; 5, 1957)

BRINK, FRANK, JR., Ph.D. Rockefeller Inst. for

Med. Research, New York City 21. *Member.* (1, 1942)

BRINK, NORMAN G., Ph.D. Merck and Co., Inc., Rahway, N. J. *Dir., Bio-Organic Chem.* (2, 1956)

BRINKHOUS, K. M., M.D. U. of North Carolina Sch. of Med., Dept. of Pathol., Chapel Hill. *Prof.* (4, 1939)

BRIODY, BERNARD A., Ph.D. Hahnemann Med. Coll., Philadelphia 2, Pa. *Prof. of Microbiol. and Dir. of Virus Lab.* (6, 1951)

BRISCOE, WILLIAM A., D.M. Bellevue Hosp., Cardio-Pulmonary Lab. C6, New York City 16. *Asst. Prof. of Med., Columbia U.* (1, 1957)

BRITTON, SYDNEY W., M.D. Route 1, Charlottesville, Va. (1R, 1925)

BROBECK, JOHN R., M.D., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Chmn., Dept. of Physiol.* (1, 1943)

BRODIE, ARNOLD F., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Assoc., Dept. of Bacteriol.* (2, 1957)

BRODIE, BERNARD B., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Chem. Pharmacol.* (2, 1940; 3, 1945)

BRODSKY, WILLIAM A., M.D. U. of Louisville Sch. of Med., Dept. of Physiol., Louisville, Ky. *Estab. Invest., Amer. Heart Assn.* (1, 1952)

BRODY, ALFRED W., M.D., D.Sc. Creighton U. Sch. of Med., Omaha 2, Nebr. *Assoc. Prof., Chair of Cardiovascular Research.* (1, 1957)

BRODY, DANIEL A., M.D. U. of Tennessee, Memphis 3. *Prof., Dept. of Med.* (1, 1953)

BRODY, THEODORE M., Ph.D. U. of Michigan, Dept. of Pharmacol., Ann Arbor. *Asst. Prof.* (3, 1953)

BROH-KAHN, ROBERT H., M.D. Labs. for Pharmaceutical Develop., Inc., Yonkers, N. Y. *Pres.* (1, 1948)

BROMILEY, REGINALD B., Ph.D. Defence Research Med. Labs., Station "K", Box 62, Toronto, Canada. *Supt., Psychol. Sociol. Wing.* (1, 1949)

BRONK, DETLEV W., Ph.D., Sc.D. Rockefeller Inst., New York City 21. *Pres.; Pres. of Natl. Acad. of Sciences.* (1, 1927)

BROOKES, MARGARET C. HESSLER, Ph.D. 5718 S. Dorchester Ave., Chicago 37, Ill. *Consultant, Chicago Heart Assn.* (5, 1935)

BROOKHART, JOHN M., Ph.D. U. of Oregon Sch. of Med., Dept. of Physiol., Portland 1. *Prof. and Head of Dept.* (1, 1946)

BROOKS, CHANDLER McCUSKEY, Ph.D. State U. of New York Coll. of Med., Dept. of Physiol., Brooklyn 3. *Prof. and Chmn.* (1, 1934; 3, 1950)

BROOKS, CLYDE, Ph.D., M.D. Station 3, Tuscaloosa, Ala. (1, 1910; 3, 1912)

BROOKS, FRANK PICKERING, M.D., Sc.D. U. of Pennsylvania Sch. of Med., Dept. of Physiol., Philadelphia 4. *Assoc. Prof.* (1, 1957)

BROOKS, MATILDA MOLDENHAUER, Ph.D. U. of California, Dept. of Physiol., Berkeley 4. *Research Assoc. in Physiol.* (1, 1923)

BROOKS, VERNON B., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (1, 1957)

BROQUIST, HARRY P., Ph.D. U. of Illinois, Dept. of Dairy Sci., Urbana. (2, 1951)

BROUHA, LUCIEN, M.D., D.Sc. Haskell Lab., E. I. duPont de Nemours & Co., Newark, Del. *Chief Physiologist.* (1, 1953)

BROUN, GORONWY OWEN, M.D. 1325 S. Grand Blvd., St. Louis, Mo. *Prof. of Int. Med., St. Louis U.* (4, 1927)

BROWN, ARNOLD L., JR., M.D. Presbyterian-St. Luke's Hosp., Chicago 12, Ill. *Instr. in Pathol., U. of Illinois.* (4, 1957)

BROWN, BARBARA BANKER, Ph.D. Riker Labs., Inc., Northridge, Calif. (3, 1951)

BROWN, CLAUDE P., M.D. 1930 Chestnut St., Philadelphia, Pa. *Practicing Clin. Pathologist.* (6, 1913)

BROWN, DAVID HENRY, Ph.D. Washington U. Med. Sch., Dept. of Biol. Chem , St. Louis 10, Mo. *Assoc. Prof.* (2, 1954)

BROWN, DUGALD E. S., Ph.D. U. of Michigan, Dept. of Zool., Ann Arbor. (1, 1932)

BROWN, EDGAR D., Pharm. D., M.D. Paynesville, Minn. *Retired.* (1R, 1907; 3R, 1909)

BROWN, ELLEN, M.D. U. of California Med. Ctr., Dept. of Med., San Francisco 22. *Assoc. Prof.* (1, 1954)

BROWN, ERNEST B., JR., Ph.D. U. of Minnesota, Dept. of Physiol., Minneapolis. *Prof.* (1, 1950)

BROWN, ETHAN ALLAN, L.R.C.P. (London). 75 Bay State Rd., Boston 15, Mass. (6, 1946)

BROWN, FRANK A., JR., Ph.D. Northwestern U., Dept. of Biol. Sci., Evanston, Ill. *Prof.* (1, 1940)

BROWN, GEORGE B., Ph.D. Sloan-Kettering Inst., New York 21. *Head, Div. Nucleoprotein Chem.; Prof. Biochem., Sloan-Kettering Div., Cornell.* (2, 1947)

BROWN, GEORGE W., Ph.D. VA Hosp., Gen. Med. Research Lab., Iowa City, Iowa. *Dir.* (1, 1955)

BROWN, GORDON CAMPBELL, Sc.D. U. of Michigan Sch. of Public Health, Ann Arbor, Mich. *Prof. of Epidemiol.* (6, 1949)

BROWN, J. H. U., Ph.D. Emory U. Sch. of Med., Dept. of Physiol., Atlanta 22, Ga. *Assoc. Prof.; Acting Chmn.* (1, 1952)

BROWN, JOHN B., Ph.D. Ohio State U., Columbus. *Prof. & Chmn., Physiol. Chem. & Pharmacol.; Dir., Inst. of Nutr. & Food Tech.* (2, 1927; 5, 1934)

BROWN, MILTON HERBERT, M.D. U. of Toronto, Toronto, Canada. *Prof. & Head, Dept. Public Health; Assoc. Dir., Sch. Hygiene.* (6, 1956)

BROWN, R. A., Ph.D. Parke, Davis & Co., Sec. #18, Detroit 32, Mich. *Dir. of Bio-Assay Div.* (5, 1946)

BROWN, RACHEL, Ph.D. 26 Buckingham Dr., Albany, N. Y. *Assoc. Biochemist, Div. of Labs. and Research, N. Y. State Dept. of Health.* (6, 1933)

BROWN, RAY K., M.D., Ph.D. New York State Dept. of Health, Albany 1. *Asst. Dir. in charge of Labs. for Biochem. and Immunol.* (2, 1956)

BROWN, ROBERT V., Ph.D. Chem. Warfare Labs., Directorate Med. Res., Army Chemical Center, Md. *Chief, Pharmacol. Br.* (1, 1945; 3, 1950)

BROWNE, J. S. L., M.D., Ph.D. McGill U., Dept. of Investigative Med., Med. Bldg. 1, Montreal, Canada. *Prof.* (1, 1934)

BROWNELL, KATHARINE A., Ph.D. Ohio State U., Dept. of Physiol., Columbus 10. *Asst. Prof.* (1, 1943)

BROZEK, JOSEF, Ph.D. U. of Minnesota, Stadium Gate 27, Minneapolis 14. *Prof., Lab. of Physiol. Hygiene.* (1, 1947; 5, 1957)

BRUES, AUSTIN M., M.D. Argonne Natl. Lab., Lemont, Ill. *Dir. of Biol. and Med. Research Div.* (1, 1940)

BRUESCH, S. R., M.D., Ph.D. U. of Tennessee, Div. of Anatomy, Memphis 3. *Prof. of Anatomy.* (1, 1949)

BRUHN, JOHN M., Ph.D. U. of Alabama Med. Ctr., Dept. of Physiol., Birmingham 3. *Prof. and Chmn.* (1, 1939)

BRUICE, THOMAS C., Ph.D. Johns Hopkins U. Sch. of Med., Dept. Physiol. Chem., Baltimore 5, Md. *Asst. Prof.* (2, 1958)

BRUNER, HARRY DAVIS, M.D., Ph.D. 9404 Byeforde Rd., Kensington, Md. *Chief, Med. Research USAEC.* (1, 1954; 3, 1945)

BRUNSCHWIG, ALEXANDER, M.D. Cornell U. Med. Coll., New York City 21. *Prof. of Clin. Surgery; tending Surgeon, Mem. Hosp.* (4, 1937)

BRUNSON, JOEL GARRETT, M.D. U. of Minnesota Sch. of Med., Minneapolis 14. *Asst. Prof. in Pathol.* (4, 1957)

BRYAN, W. RAY, Ph.D. 4405 Bywood Lane, Bethesda, Md. *Biologist, Natl. Cancer Inst., NIH.* (1, 1934; 4, 1940; 6, 1954)

BRYANT, HAROLD H., Ph.D. Hynson Westcott and Dunning, Inc., Baltimore 1, Md. *Pharmacologist.* (3, 1958)

BUCHANAN, DONALD L., M.D. VA Hosp., West Haven 16, Conn. *Chief, Radioisotope Serv.* (2, 1954)

BUCHANAN, JOHN M., Ph.D. Massachusetts Inst. of Tech., Div. of Biochem., Cambridge. *Prof. & Head.* (2, 1949)

BUCHBINDER, LEON, Ph.D. Dept. of Health, 125 Worth St., New York City. *Asst. Dir., Bur. of Labs.* (6R, 1934)

BUCHBINDER, WILLIAM C., M.D. 1893 Sheridan Rd. Highland Park, Ill. *Assoc. Prof., Med., Northwestern U. Med. Sch.* (1, 1940)

BUCHER, NANCY L. R., M.D. Massachusetts Gen. Hosp., Huntington Labs., Boston 14. *Assoc. Biologist; Res. Assoc., Med., Harvard.* (1, 1955)

BUCKLEY, NANCY MARGARET, M.D. Albert Einstein Coll. of Med., Dept. of Physiol., New York City 61. *Asst. Prof.* (1, 1957)

BUCKNER, G. DAVIS, Ph.D. Kentucky Agric. Expt. Sta., Lexington. *In charge of Animal Nutrition (Emer.).* (2R, 1920)

BUCY, PAUL C., M.D. 5830 Stony Island Ave., Chicago 37, Ill. *Prof. of Surgery, Northwestern U. Med. Sch.* (1, 1933)

BUDDINGH, G. JOHN, M.D. Louisiana State U. Sch. of Med., New Orleans 12. *Prof. of Microbiol.; Head of Dept.* (4, 1940)

BUDY, ANN MARIE, Ph.D. U. of Chicago, Chicago 37, Ill. *Research Assoc. (Instr.), Physiol. & Pharmacol.* (3, 1958)

BUEDING, ERNEST, M.D. Louisiana State U. Sch. of Med., New Orleans 12. *Prof. of Pharmacol.; Chmn. of Dept.* (2, 1946; 3, 1949)

BUELL, MARY V., Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Prof.* (2, 1921)

BUESCHER, EDWARD LOUIS, M.D. Walter Reed Army Inst. of Research, Washington 12, D. C. *Chief, Dept. of Virus Diseases.* (6, 1957)

BUETTNER, K., Ph.D. U. of Washington, Dept. of Meteorol., Seattle 5. *Prof. of Meteorol. and Research Physiologist.* (1, 1955)

BUGHER, JOHN C., M.D. The Rockefeller Fndn., 49 W. 49th St., New York City 20. *Dir. for Med. Education and Public Health.* (4, 1935)

BUKANTZ, SAMUEL C., M.D. 3447 W. 19th Ave., Denver 4, Colo. *Med. & Res. Dir., JNHAC; Assoc. Clin. Prof. Med., U. of Colo.* (6, 1943)

BULATAO, EMILIO, M.D. U. of the Philippines, Manila, P. I. *Prof. of Physiol. and Biochem.* (1R, 1924)

BULL, HENRY B., Ph.D. State U. of Iowa, Dept. of Biochem., Iowa City. *Prof. and Head of Dept.* (2, 1937)

BULLOCK, THEODORE H., Ph.D. U. of California, Los Angeles 24. *Prof. of Zool.* (1, 1948)

BUNDE, CARL A., M.D., Ph.D. Pitman-Moore Co., Indianapolis 6, Ind. *Vice-Pres., Research.* (1, 1943)

BUNKER, JOHN P., M.D. Massachusetts Gen. Hosp., Dept. of Anesthesia, Boston 14. *Asst. Clin. Prof. Anesthesia, Harvard Med. Sch.* (3, 1954)

BUNNEY, WILLIAM EDWARD, Ph.D. E. R. Squibb & Sons, Div., Olin Mathieson Chem. Corp., New Brunswick, N. J. *Vice Pres.; Dir. Mfg.* (6, 1931)

BUNTING, CHARLES H., M.D. 50 Broad Ripple Dr., Princeton, N. J. *Prof. Emer. of Pathol., U. of Wisconsin.* (4R, 1913)

BUNZELL, H. H., Ph.D. Box 44, New York City 1. *Dir. of Bunzell Labs.* (2R, 1908)

BURBRIDGE, THOMAS N., M.D., Ph.D. U. of California Sch. of Med., Dept. of Pharmacol., San Francisco 22. *Asst. Prof.* (3, 1957)

BURCH, HELEN B., Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Assoc. Prof.* (2, 1956; 5, 1958)

BURCHELL, HOWARD B., M.D., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant in Med.; Prof. in Med., Mayo Fndn.* (1, 1942)

BURCHENAL, JOSEPH H., M.D. Sloan-Kettering Inst., Cornell U. Med. Coll., New York City 21. *Prof. of Med.* (5, 1954)

BURDICK, H. O., Sc.D. Alfred U., Dept. of Biol., Alfred, N. Y. *Prof. and Chmn. of Dept.* (1, 1940)

BURDON, KENNETH L., Ph.D. Baylor U. Coll. of Med., Dept. of Microbiol., Houston, Tex. *Prof. & Chmn. of Dept.* (6, 1936)

BURGEN, A. S. V., M.B., MRCP. McGill U., Dept. of Physiol., Montreal, Canada. *Prof.* (1, 1950; 3, 1950)

BURGISON, RAYMOND M., Ph.D. U. of Maryland Sch. of Med., Baltimore 1. *Assoc. Prof.* (3, 1958)

BURK, DEAN, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head Chemist, PHS.* (2, 1939)

BURKE, BERTHA S., M.A. Harvard Sch. of Public Health, Boston 15, Mass. *Assoc. Prof. of Maternal and Child Nutrition.* (5, 1952)

BURKE, JOHN C., Ph.D. E.R. Squibb & Sons, Div. of Pharmacol., New Brunswick, N. J. *Dept. Head.* (3, 1953)

BURLINGTON, HAROLD, Ph.D. U. of Cincinnati Coll. of Med., Dept. of Physiol., Cincinnati 19,

Ohio. *Asst. Prof.* (1, 1958)

BURN, J. HAROLD, M.D. Oxford U., Oxford, England. *Prof. in charge of Dept. of Pharmacol.* (3, 1954)

BURNS, B. DELISLE. McGill U., Dept. of Physiol., Montreal, Canada. *Assoc. Prof.* (1, 1954)

BURNS, EDWARD L., M.D. Mercy Hosp., Toledo, Ohio. *Pathologist.* (4, 1939)

BURNS, JOHN J., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Asst. Chief, Lab. of Chem. Pharmacol.* (2, 1956; 3, 1954)

BURR, GEORGE O., Ph.D. Expt. Sta., H.S.P.A., Honolulu, Hawaii. *Head, Dept. of Biochem. and Physiol.* (2, 1928; 5, 1933)

BURR, WILLIAM W., JR., Ph.D. U. of Texas, Southwestern Med. Sch., Dallas. *Assoc. Prof. of Biochem.* (2, 1958)

BURRIS, ROBERT H., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Prof.* (2, 1946)

BURROUGHS, WISE, Ph.D. Iowa State Coll., Dept. of Animal Husbandry, Ames. *Prof.* (5, 1951)

BURROWS, WILLIAM, Ph.D. U. of Chicago, Dept. of Microbiol., Chicago 37, Ill. *Prof.* (6, 1947)

BURSTONE, M. S., D.D.S. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Dental Surgeon.* (4, 1955)

BURTON, ALAN C., Ph.D. U. of Western Ontario Med. Sch., Dept. of Biophysics, London, Canada. *Prof. and Head of Dept.* (1, 1937)

BURTON, ROBERT MAIN, Ph.D. Washington U. Sch. of Med., Dept. of Pharmacol., St. Louis 10, Mo. *Asst. Prof.* (2, 1958)

BUSCH, HARRIS, M.D., Ph.D. U. of Illinois Coll. of Med., Chicago 12, Ill. *Assoc. Prof. of Pharmacol.* (2, 1955; 3, 1957)

BUSH, MILTON T., Ph.D. Vanderbilt U. Sch. of Med., Nashville 4, Tenn. *Assoc. Prof. of Pharmacol.* (3, 1938)

BUSKIRK, ELSWORTH R., Ph.D. Natl. Inst. of Arthritis & Metabolic Dis., NIH, Bethesda 14, Md. *Physiologist-in-Charge, Metabolic Chamber.* (1, 1958)

BUSSARD, ALAIN, Ph.D. Pasteur Inst., Paris, France. *Chef de Laboratoire.* (6, 1952)

BUTLER, G. C., Ph.D. Atomic Energy of Canada, Ltd., Biol. & Health Physics Div., Chalk River, Ont., Canada. *Dir.* (2, 1949)

BUTLER, LILLIAN C., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Guest Worker, Sec. on Endocrinol.* (5, 1958)

BUTLER, THOMAS C., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Prof. of Pharmacol.* (3, 1938)

BUTT, HUGH R., M.D. Mayo Clinic, 200 First St., S. W., Rochester, Minn. *Consultant in Med.; Prof. of Med., Mayo Fndn.* (5, 1942)

BUTTS, JOSEPH S., Ph.D. Oregon State Coll., Corvallis. *Prof. of Biochem. and Head of Agric. Chem.* (2, 1936; 5, 1936)

BYERRUM, RICHARD U., Ph.D. Michigan State U., Kedzie Chem. Lab., East Lansing. *Prof. of Chem.* (2, 1954)

BYERS, SANFORD O., Ph.D. 1600 Divisadero St., San Francisco 15, Calif. *Research Biochemist, Mount Zion Hosp.* (1, 1949)

CAFRUNY, EDWARD JOSEPH, Ph.D. U. of Michigan Med. Sch., Dept. of Pharmacol., Ann Arbor. *Instr.* (3, 1958)

CAHEN, RAYMOND L., Ph.D., M.D. Laboratoires Lafon, 1, rue georges Médéric, Maisons-Alfort (Seine), France. *Research Dir.* (3, 1949)

CAHILL, WILLIAM M., Ph.D., M.D. Southern California Edison Co., 501 S. Marengo, Alhambra. *Physician & Surgeon, Med. Dept.* (2, 1940)

CAJORI, FLORIAN A., Ph.D. U. of Colorado Med. Sch., Denver 20. *Assoc. Prof. of Biochem.* (2, 1922; 5, 1933)

CALDWELL, MARY L., Ph.D. Columbia U., New York City 27. *Prof. of Chem.* (2, 1924; 5, 1933)

CALESNICK, BENJAMIN, M.D. 1036 S. 54th St., Philadelphia 43, Pa. *Asst. Prof. in Human Pharmacol., Habnemann Med. Coll.* (3, 1951)

CALHOON, THOMAS BRUCE, Ph.D. Med. Coll. of South Carolina, Dept. of Physiol., Charleston. *Asst. Prof.* (1, 1957)

CALLOWAY, DORIS H., Ph.D. QM Food and Container Inst., Nutrition Br., 1819 W. Pershing Rd., Chicago 9, Ill. *Nutritionist.* (5, 1955)

CALLOWAY, NATHANIEL OGLESBY, Ph.D., M.D. Med. Associates of Chicago 3233 S. Parkway, Chicago 16, Ill. (3, 1945)

CALVIN, D. BAILEY, Ph.D. U. of Texas Med. Br., Galveston. *Prof. of Biol. Chem. and Dean of Students.* (1, 1934; 2, 1939)

CALVIN, MELVIN, Ph.D. U. of California, Berkeley 4. *Prof. of Chem.* (2, 1949)

CAMIEN, MERRILL N., Ph.D. U. of California at Los Angeles, Dept. of Chem., Los Angeles 24. *Assoc. Research Chemist.* (2, 1953)

CAMINOPETROS, JOHN, M.D. 61 Emm. Benaki Str., and A. Metaxa 17, Athens, Greece. (6, 1946)

CAMP, WALTER J. R., M.D., Ph.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Pharmacol. & Toxicol.; State Toxicologist.* (3, 1926)

CAMPBELL, BERRY, Ph.D. U. of Minnesota, Minneapolis 14. *Prof. of Anatomy.* (1, 1945)

CAMPBELL, DAN H., Ph.D. California Inst. of Tech., Div. of Chem. & Chem. Engin., Pasadena. *Prof. of Immunochem.* (6, 1938)

CAMPBELL, H. LOUISE, Ph.D. 916 Windsor Ave., Windsor, Conn. (5R, 1933)

CAMPBELL, J. A., Ph.D. Dept. Natl. Health & Welfare, Food & Drug Div., Ottawa, Canada. *Chief, Vitamin and Nutr. Lab.* (5, 1950)

CAMPBELL, JAMES, Ph.D. U. of Toronto, Toronto, Canada. *Assoc. Prof. of Physiol.* (1, 1943)

CAMPBELL, L. LEON, JR., Ph.D. State Coll. of Washington, Lab. of Food Microbiol., Pullman. *Assoc. Prof. & Assoc. Bacteriologist.* (2, 1957)

CAMPBELL, WALTER RUGGLES, M.D. 101 Douglas Dr., Toronto, Canada. *Assoc. Prof. of Med., U. of Toronto.* (2R, 1922)

CANELLAKIS, E. S., Ph.D. Yale U. Sch. of Med., Dept. of Pharmacol., New Haven, Conn. *Asst. Prof.* (2, 1956)

CANN, JOHN R., Ph.D. U. of Colorado Sch. of Med., Denver 20. *Assoc. Prof. of Biophysics.* (6, 1955)

CANNAN, R. KEITH, D.Sc. Natl. Research Council, 2101 Constitution Ave., Washington 25, D. C. *Chmn., Div. of Med. Sciences.* (2, 1931)

CANNON, PAUL R., M.D., Ph.D. U. of Chicago Clinics, Chicago, Ill. *Emer. Prof. of Patbol.* (4, 1930; 6, 1929)

CANTAROW, ABRAHAM, M.D. Jefferson Med. Coll.,

Philadelphia 7, Pa. *Prof. of Biochem. and Head of Dept.* (1, 1932; 3, 1935)

CANTONI, G. L., M.D. Natl. Inst. of Mental Health, NIH, Bethesda 14, Md. *Chief, Lab. of Cellular Pharmacol.* (2, 1953; 3, 1945)

CANTRELL, WILLIAM F., Ph.D. U. of Louisville, Louisville 2, Ky. *Assoc. Prof. of Pharmacol.* (3, 1954)

CANZANELLI, ATTILIO, M.D. Tufts U. Sch. of Med. Boston 11, Mass. *Prof. of Physiol.* (1, 1934)

CAPPS, ROBERT T., M.D., Ph.D. Parkland Mem. Hosp., Dallas 35, Tex. *Asst. Prof. of Anesthesia.* (3, 1951)

CAPUTTO, RANWEL, M.D. Oklahoma Med. Research Fndn., Oklahoma City 4. *Head, Biochem.* (2, 1955)

CARLSON, LOREN D., Ph.D. U. of Washington Sch. of Med., Dept. of Physiol. and Biophysics, Seattle 5. (1, 1945)

CARMICHAEL, EMMETT B., Ph.D. Med. Coll. of Alabama & Sch. of Dent., Dept. of Biochem., Birmingham 3. *Prof. & Chmn. of Dept.* (1, 1931; 2, 1946)

CARMICHAEL, LEONARD, Ph.D., Sc.D. Smithsonian Institution, Washington 25, D. C. *Secy. (Dir.).* (1, 1937)

CARNES, WILLIAM H., M.D. Salt Lake Gen. Hosp., Salt Lake City 15, Utah. *Prof. of Pathol., U. of Utah.* (4, 1951)

CARPENTER, FRANK G., Ph.D. Dartmouth Med. Sch., Dept. of Physiol. Science, Hanover, N. H. *Asst. Prof.* (1, 1956)

CARPENTER, FREDERICK H., Ph.D. U. of California, Dept. of Biochem., Berkeley 4. *Assoc. Prof.* (2, 1949)

CARPENTER, THORNE M., Ph.D. 142 Birch Tree Dr., Westwood, Mass. (1R, 1915; 2R, 1909; 5R, 1933)

CARR, C. JELLEFF, Ph.D. Natl. Insts. of Health, Psychopharmacol. Serv. Ctr., Bethesda 14, Md. *Sr. Research Pharmacologist.* (3, 1940)

CARR, CHARLES W., Ph.D. U. of Minnesota, Dept. of Physiol. Chem., Minneapolis 14. *Asst. Prof.* (2, 1957)

CARR, EDWARD A., JR., M.D. U. of Michigan, Ann Arbor. *Assoc. Prof. of Pharmacol.; Assoc. Prof. of Int. Med.* (3, 1957)

CARR, JESSE L., M.D. San Francisco Hosp., San Francisco, Calif. *Clin. Prof. of Pathol.* (4, 1940)

CARRERA, GUILLERMO M., M.D. Ochsner Fndn. Hosp., 1516 Jefferson Hwy., New Orleans, La. (4, 1951)

CARRICK, C. W., M.S.A. Purdue U., Lafayette, Ind. *Prof. of Poultry Sci.* (5, 1954)

CARROLL, KENNETH K., Ph.D. U. of Western Ontario, Collip Med. Res. Lab., London, Canada. *Assoc. Prof.* (2, 1958)

CARRUTHERS, CHRISTOPHER, Ph.D. Roswell Park Mem. Inst., Buffalo 3, N. Y. *Assoc. Cancer Research Scientist (Biochem.).* (2, 1948)

CARSON, STANLEY F., Ph.D. Oak Ridge Natl. Lab., Biol. Div., P. O. Box Y, Oak Ridge, Tenn. *Prin. Biochemist.* (2, 1952)

CARSTEN, MARY E., Ph.D. U. of California Med. Ctr., Dept. of Physiol. Chem., Los Angeles 24. *Asst. Res. Physiol. Chemist.* (2, 1958)

CARTER, CHARLES E., M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Prof. of Pharmacol.* (2, 1950)

CARTER, EARL T., M.D., Ph.D. Ohio State U., Dept. of Physiol., Columbus 10. *Asst. Prof.* (1, 1958)

CARTER, HERBERT E., Ph.D. U. of Illinois, 108 Noyes Lab., Urbana. *Prof. of Biochem. and Head of Dept.* (2, 1937; 5, 1941)

CARTER, JOHN R., M.D. State U. of Iowa Coll. of Med., Dept. of Pathol., Iowa City. *Prof. of Pathol.* (4, 1951)

CARTLAND, GEORGE F., Ph.D. The Upjohn Co., Kalamazoo, Mich. *Dir. of Sci. Relations.* (2, 1936)

CARTWRIGHT, G. E., M.D. Salt Lake Gen. Hosp., Salt Lake City 15, Utah. *Prof. of Med., U. of Utah.* (5, 1951)

CARY, CHARLES A., S.B. 141 N. W. 100th Terr., Miami Shores, Fla. (2R, 1920)

CASARETT, GEORGE W., Ph.D. U. of Rochester Med. Ctr., Rochester 20, N. Y. *Asst. Prof., Radiation Biol.; Scientist, AE Project.* (4, 1957)

CASAS, CARMEN B., Ph.D. U. of Puerto Rico Sch. of Tropical Med., San Juan 22. *Assoc. Prof. of Physiol.* (1, 1952)

CASEY, ALBERT EUGENE, M.D. 2236 Highland Ave., Birmingham 9, Ala. *Pathologist & Dir. of Labs., Baptist Hosp.; Prof. Pathol., U. of Ala.* (4, 1933)

CASH, JAMES ROBERT, M.D. University Hosp., Charlottesville, Va. *Prof. of Pathol., U. of Virginia Sch. of Med.* (4, 1924)

CASSEL, WILLIAM A., Ph.D. Hahnemann Med. Coll., Philadelphia 2, Pa. *Asst. Prof. of Microbiol.* (6, 1958)

CASSELMAN, W. G. BRUCE, Ph.D., M.D. New York State Psychiatric Inst., Dept. Neuropathol., New York City 32. *Research Histochemist.* (1, 1957)

CASSIDY, HAROLD G., Ph.D. Yale U., Dept. of Chem., New Haven, Conn. *Prof.* (2, 1949)

CASTER, W. O., Ph.D. U. of Minnesota, 227 Millard Hall, Minneapolis 14. *Asst. Prof., Physiol. Chem.* (2, 1956)

CASTLE, EDWARD S., Ph.D. Harvard U. Biol. Labs., Cambridge 38, Mass. *Prof. of Physiol.* (1, 1934)

CASTLE, WILLIAM B., M.D. Boston City Hosp., Boston, Mass. *George Richards Minot Prof. of Med., Harvard U.; Dir., Thorndike Mem. Lab.* (4, 1942)

CASTLEMAN, BENJAMIN, M.D. Massachusetts Gen. Hosp., Dept. of Pathol., Boston. *Chief; Clin. Prof. of Pathol., Harvard Med. Sch.* (4, 1954)

CATCHPOLE, HUBERT RALPH, Ph.D. U. of Illinois Coll. of Med., Chicago 12. *Research Assoc. Prof. of Pathol.* (1, 1941)

CATRON, DAMON, Ph.D. Iowa State Coll., Dept. of Animal Husbandry, Ames. *Prof.* (5, 1953)

CATRON, LLOYD, M.D. Akron City Hosp., Dept. of Pathol., Akron, Ohio. *Dir.* (4, 1939)

CATTELL, McKEEN, Ph.D., M.D. Cornell U. Med. Coll., New York City 21. *Prof. of Clin. Pharmacol.* (1, 1923; 3, 1924)

CAVALIERI, LIEBE F., Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc. Prof. Biochem., Sloan-Kettering Div., Cornell Med. Coll.* (2, 1953)

CAVANAUGH, DANIEL J., Ph.D. Southeast Louisiana Hosp., Mandeville. *Biochemist.* (3, 1957)

CAVELTI, PHILIP A., M.D. Medical Plaza, Welch at Pasteur, Palo Alto, Calif. (6, 1947)

CAVERT, H. MEAD, M.D., Ph.D. U. of Minnesota Med. Sch., Dept. of Physiol., Minneapolis 14. *Asst. Prof.* (1, 1954)

CAZORT, RALPH JERRY, M.D. Meharry Med. Coll., Nashville 8, Tenn. *Assoc. Prof. of Pharmacol.* (3, 1951)

CEDERQUIST, DENA C., Ph.D. Michigan State U., Coll. of Home Econ., East Lansing. *Prof. of Foods & Nutrition.* (5, 1949)

CEITHAML, JOSEPH J., Ph.D. U. of Chicago, Chicago 37, Ill. *Prof. of Biochem.; Dean of Students in Biol. Sci.* (2, 1950)

CELMER, WALTER D., Ph.D. Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y. *Antibiotics Res. Supervisor.* (2, 1958)

CERECEDO, LEOPOLD R., Ph.D. Fordham U., Dept. of Chem., New York City. *Prof. of Biochem.* (2, 1931; 5, 1945)

CHADWICK, LEIGH EDWARD, Ph.D. U. of Illinois, Dept. of Entomol., Urbana. *Dept. Head.* (1, 1944)

CHAIKOFF, I. L., Ph.D., M.D. U. of California, Berkeley 4. *Prof. of Physiol.* (1, 1932)

CHALKLEY, HAROLD W., Ph.D. 4609 Highland Ave., Bethesda 14, Md. (1R, 1932)

CHAMBERLAIN, W. EDWARD, M.D. 152 Van Winkle Dr., Falls Church, Va. *Spec. Asst. for Atomic Med., and Assoc. Dir., Res. Serv. VA.* (1, 1948)

CHAMBERS, ALFRED H., Ph.D. U. of Vermont Coll. of Med., Dept. of Physiol. & Biophysics, Burlington. *Assoc. Prof.* (1, 1946)

CHAMBERS, EDWARD L., M.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Assoc. Prof. of Physiol.* (1, 1956)

CHAMBERS, LESLIE ADDISON, Ph.D. County Los Angeles Air Pollution Control Dist., Los Angeles 13, Calif. *Dir. Research.* (1, 1940; 6, 1948)

CHAMBERS, W. W., Ph.D. U. of Pennsylvania, Sch. of Med., Dept. of Anatomy, Philadelphia 4. *Prof.* (1, 1950)

CHAMBERS, WILLIAM H., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Chief, Physiol. Div.* (1, 1924; 5, 1933)

CHANCE, BRITTON, Ph.D., D.Sc. U. of Pennsylvania, Johnson Fndn., Philadelphia 4. *Prof. and Dir. of Fndn.* (1, 1950; 2, 1950)

CHANDLER, JOSEPH P., Ph.D. U. of Michigan Med. Sch., Room G455, Univ. Hosp., Ann Arbor. *Assoc. Prof. of Biol. Chem.* (2, 1944; 5, 1944)

CHANG, HSIANG-TUNG, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc.* (1, 1954)

CHANG, MEI-LING WU, Ph.D. U. of Illinois, Div. of Nutrition, Dept. of Animal Sci., Urbana. *Res. Assoc. in Animal Nutrition.* (5, 1958)

CHANG, MIN CHUEH, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Research Assoc.* (1, 1946)

CHANGUS, GEORGE W., M.D. Northwestern U. Sch. of Med., Chicago 11, Ill. (4, 1951)

CHANUTIN, ALFRED, Ph.D. U. of Virginia Sch. of Med., Biochem. Lab., Charlottesville. *Prof. of Biochem.* (2, 1925)

CHAPIN, JOHN LADNER, Ph.D. U. of Colorado Med. Sch., Dept. of Physiol., Denver 20. *Asst. Prof.* (1, 1957)

CHAPMAN, LORING FREDERICK, Ph.D. Cornell U. Med. Coll., New York City 21. *Research Fellow.* (1, 1957)

CHARALAMPOUS, FRIXOS C., M.D., Sc.D. U. of Pennsylvania Med. Sch., Dept. of Chem., Philadelphia 4. *Assoc. Prof. of Biochem.* (2, 1955)

CHARGAFF, ERWIN, Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Prof. of Biochem.* (2, 1935)

CHARIPPER, HARRY ADOLPH, Ph.D. Washington Square Coll. of Arts and Sci., New York City. *Prof. and Head, Dept. of Biol.* (1, 1941)

CHARNEY, JESSE, M.S. Merck Sharp & Dohme Research Labs., West Point, Pa. *Research Assoc.* (2, 1954)

CHASE, AURIN M., Ph.D. Princeton U., Dept. of Biol., Princeton, N. J. *Assoc. Prof.* (1, 1939; 2, 1957)

CHASE, HAROLD F., M.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Clin. and Research Prof.* (3, 1944)

CHASE, MERRILL W., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (6, 1938)

CHASIS, HERBERT, M.D., Med. Sc.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (1, 1941)

CHATFIELD, CHARLOTTE, B.S. 4 El Sereno Dr., San Carlos, Calif. (5, 1941)

CHATFIELD, PAUL OAKES, M.D. Jefferson Med. Coll., Dept. of Neurol., Philadelphia 7, Pa. *Asst. Prof. of Neurophysiol.* (1, 1950)

CHATTERJEE, HEMENDRA NATH, M.D. 9 Romes Mitter Rd., Bhowanipur, Calcutta 25, India. (6, 1948)

CHEEVER, FRANCIS SARGENT, M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Prof. of Microbiol.* (6, 1950)

CHELDELIN, VERNON H., Ph.D. Oregon State Coll., Corvallis. *Prof. of Chem.; Dir., Sci. Research Inst.* (2, 1947; 5, 1946)

CHEN, GRAHAM, Sc.D., M.D. Parke, Davis & Co., Detroit 32, Mich. *Lab. Dir.; Research Pharmacologist.* (3, 1944)

CHEN, JAMES YU-PING, M.D. Marquette U. Sch. of Med., Dept. of Pharmacol., Milwaukee 3, Wis. *Prof.* (3, 1951)

CHEN, K. K., Ph.D., M.D. Lilly Research Labs., Indianapolis 6, Ind. *Dir. of Pharmacol. Research.* (1, 1929; 3, 1925)

CHENEY, RALPH H., Sc.D. Brooklyn Coll., Biol. Dept., Brooklyn 10, N.Y. *Prof.* (3, 1934)

CHENG, AMBER L. S., Ph.D. U. of California Med. Ctr., Dept. of Surgery, Los Angeles 24. *Research Assoc.* (5, 1957)

CHENOWETH, MAYNARD B., M.D. The Dow Chemical Co., Biochem. Research Lab., Midland, Mich. (3, 1945)

CHERNICK, SIDNEY S., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Scientist, PHS.* (2, 1954)

CHESLEY, LEON C., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Prof. of Obstetrics & Gynecol.* (1, 1949)

CHESNEY, ALAN M., M.D. Johns Hopkins Hosp., Bal-

timore 5, Md. *Dean Emer., Med. Faculty; Assoc. Prof. Emer. of Med., Johns Hopkins U.* (4, 1925)

CHILD, GEORGE P., Ph.D., M.D. Roselle Park, N. J. (3, 1949)

CHIN, YIN-CH'ANG, Ph.D. 1268 Sixth Ave., San Francisco, Calif. *Research Fellow, U. of Calif.; Asst. Prof., Peking Union Med. Coll.* (3, 1950)

CHINARD, F. P., M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. Physiol. Chem. & Med.; Asst. Chief Med., City Hosps.* (2, 1951)

CHINN, HERMAN I., Ph.D. Office of Sci. Res., Air Res. & Develop. Command, Washington 25, D. C. *Biochemist.* (1, 1950; 3, 1951)

CHOPRA, R. N., M.D., Sc.D. Drug Research Lab., Canal Rd., Jammu-Tawi, Kashmir, India. *Dir.* (3H, 1938)

CHOW, BACON F., Ph.D. Johns Hopkins U., Sch. of Hygiene, Baltimore 5, Md. *Assoc. Prof. Biochem.* (2, 1940; 5, 1948; 6, 1944)

CHOW, KAO LIANG, Ph.D. U. of Chicago, Dept. of Physiol., Chicago 37, Ill. *Asst. Prof.* (1, 1958)

CHRISTENSEN, HALVOR N., Ph.D. U. of Michigan, Dept. of Biol. Chem., Ann Arbor. *Prof. and Head of Dept.* (2, 1947)

CHRISTENSEN, L. ROYAL, Ph.D. New York U. Coll. of Med., Dept. of Microbiol., New York City 16. *Assoc. Prof.; Dir., Berg Inst.* (6, 1942)

CHRISTIAN, JOHN J., Sc.D. Naval Med. Research Inst., Bethesda 14, Md. *Physiologist, Exptl. Med.* (4, 1955)

CHRISTIE, R. V., M.D., D.Sc. Royal Victoria Hosp., Montreal, Canada. *Physician-in-Chief; Prof. Med. & Chmn. of Dept., McGill U.* (1, 1957)

CHRISTIE, ROBERT W., M.D. Natl. Insts. of Health, Clin. Ctr., Bethesda 14, Md. *Resident in Clin. Pathol.* (4, 1958)

CHRISTMAN, ADAM A., Ph.D. U. of Michigan Med. Sch., Dept. of Biol. Chem., Ann Arbor. *Prof.* (2, 1929)

CHURNEY, LEON, Ph.D. Louisiana State U. Sch. of Med., New Orleans 12. *Assoc. Prof. of Physiol.* (1, 1953)

CHUSID, JOSEPH G., M.D. St. Vincent's Hosp., Neurol. Div., New York City 11. *Attending Neurologist.* (1, 1951)

CIFONELLI, J. ANTHONY, Ph.D. La Rabida- U. of Chicago Inst., Chicago 49, Ill. *Research Assoc.* (2, 1958)

CIZEK, LOUIS JOSEPH, M.D. Columbia U. Coll. of P. & S., Dept. of Physiol., New York City 32. *Assoc. Prof.* (1, 1951)

CLAMANN, HANS G., M.D. USAF Sch. of Aviation Med., Dept. of Physiol. & Biophysics, Randolph AFB, Tex. *Res. Physiologist.* (1, 1953)

CLARE, MARGARET H., M.A. Washington U. Sch. of Med., Lab. of Neurophysiol., St. Louis 10, Mo. *Asst. Prof.* (1, 1955)

CLARK, BYRON B., Ph.D. Mead Johnson & Co., Research Labs., Evansville 21, Ind. *Dir., Div. of Pharmacol. & Chemotherapy.* (3, 1940)

CLARK, ELIOT R., M.D. U. of Pennsylvania, Philadelphia 4. *Prof. Emer. of Anatomy.* (1R, 1919)

CLARK, ERNEST D., Ph.D. 4904 Princeton Way, Seattle 15, Wash. (2R, 1912)

CLARK, GEORGE, Ph.D. U. of Buffalo, Dept. of Physiol., Buffalo 14, N. Y. *Assoc. Prof.* (1, 1943)

CLARK, GUY W., Ph.D. 5330 Wild Horse Valley Rd., Napa, Calif. (2R, 1922)

CLARK, IRWIN, Ph.D. Merck Inst. for Therap. Research, Rahway, N. J. *Head, Isotope Dept.* (2, 1956)

CLARK, JANET HOWELL, Ph.D. 708 Cedarcroft Rd., Baltimore, Md. *Research Assoc., Dept. of Environ. Med., Johns Hopkins U.* (1R, 1922)

CLARK, JOHN K., M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Assoc. Prof. Med.; Dir. Res., Smith Kline & French Labs.* (1, 1953)

CLARK, LELAND C., JR., Ph.D. U. of Alabama Med. Ctr., Dept. of Surgery, Birmingham 3. *Assoc. Prof. of Biochem.* (1, 1953)

CLARK, PAUL F., Ph.D. U. of Wisconsin Med. Sch. Madison. *Prof. Emer. of Med. Microbiol.* (6R, 1928)

CLARK, ROBERT T., JR., Ph.D. USAF Sch. of Aviation Med., Dept. of Physiol. & Biophysics, Randolph AFB, Tex. *Head.* (1, 1953)

CLARK, W. G., Ph.D. VA Ctr., Los Angeles 25, Calif. *Chief, Pharmacol.-Physiol. Sec.; Assoc. Prof., Physiol. Chem., U. Calif.* (1, 1942; 3, 1949)

CLARK, WILLIAM MANSFIELD, Ph.D. Johns Hopkins U., Baltimore 18, Md. *DeLamar Prof. Emer. Physiol. Chem.; Res. Prof. Chem.* (2R, 1920)

CLARKE, DONALD A., Ph.D. Sloan-Kettering Inst., New York City 21. (3, 1952)

CLARKE, HANS THACHER, D.Sc. (London). Yale U., Dept. of Biochem., New Haven, Conn. *Lect. in Biochem.* (2, 1929)

CLARKE, MIRIAM F., Ph.D. Woman's Med. Coll. of Pennsylvania, Physiol. Chem. Dept., Philadelphia 29. *Assoc. Prof.* (5, 1958)

CLARKE, ROBERT W. Walter Reed Army Inst. of Research, Washington 12, D. C. *Chief, Dept. of Surgical Physiol.* (1, 1936)

CLEGG, ROBERT E., Ph.D. Kansas State Coll., Manhattan. *Prof. of Chem.* (2, 1957)

CLEGHORN, ROBERT ALLEN, M.D., D.Sc. McGill U., Dept. of Psychiatry, Montreal, Canada. *Assoc. Prof.* (1, 1937)

CLEMENTE, CARMINE D., Ph.D. U. of California Sch. of Med., Dept. of Anatomy, Los Angeles 24. *Asst. Prof.* (1, 1957)

CLEMMONS, JACKSON J., Ph.D. Western Reserve U. Inst. of Pathol., Cleveland, Ohio. *Fellow in Pathol.* (4, 1956)

CLOWES, GEORGE HENRY ALEXANDER, Ph.D. Eli Lilly & Co., Indianapolis 6, Ind. *Dir. of Research Emer.* (2R, 1914; 6R, 1919)

CLUFF, LEIGHTON E., M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. in Med.; Head, Div. of Immunol., Johns Hopkins U.* (6, 1954)

COBURN, ALVIN F., M.D. 14 Ludlow Dr., Chappaqua, N. Y. (6, 1948)

COCA, ARTHUR F., M.D. 425 Grant Ave., Oradell, N. J. (6H, 1916)

COCHIN, JOSEPH, M.D., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Clin. Pharmacologist.* (3, 1955)

COCHRAN, K. W., Ph.D. U. of Michigan, Dept. of Epidemiol., Ann Arbor. *Asst. Prof.* (3, 1952; 6, 1957)

CODE, CHARLES F., Ph.D., M.D. Mayo Fndn., Rochester, Minn. *Prof. of Physiol.* (1, 1939;

3, 1950)

COFFEY, JULIA M., A.B. New York State Dept. of Health, Div. of Labs. & Research, Albany. *Assoc. Bacteriologist.* (6, 1937)

COGHILL, ROBERT D., Ph.D. Cancer Chemotherapy Natl. Serv. Ctr., NIH, Bethesda 14, Md. *Dep. Chief for Indust. Research.* (2, 1932)

COHEN, HERMAN, Ph.D. Princeton Labs., Princeton, N. J. *Research Assoc.* (1, 1955)

COHEN, JONATHAN, M.D. Children's Med. Ctr., Boston 15, Mass. *Asst. Orthopedic Surgeon and Asst. Pathologist.* (4, 1956)

COHEN, JULIUS J., M.D. U. of Cincinnati Coll. of Med., Depts. of Physiol. & Med., Cincinnati 19, Ohio. *Asst. Prof.* (1, 1957)

COHEN, LEONARD A., Ph.D. U. of Pittsburgh Sch. of Med. Dept. of Physiol. & Pharmacol., Pittsburgh 13, Pa. *Asst. Prof.* (1, 1956)

COHEN, PHILIP P., Ph.D., M.D. U. of Wisconsin, Med. Sch., Madison 6. *Prof. of Physiol. Chem.* (2, 1941)

COHEN, SAUL L., Ph.D. 52 Meadowbrook Dr., Apt. 4, Toronto 19, Canada. (2, 1948)

COHEN, SEYMOUR S., Ph.D. U. of Pennsylvania Sch. of Med., Dept. of Biochem., Philadelphia 4, Pa. *Prof. of Biochem. and of Pediatrics.* (2, 1946)

COHEN, SOPHIA M., B.S. New York State Dept. of Health, Div. of Labs. & Research, Albany 1. *Assoc. Bacteriologist.* (6, 1938)

COHEN, STANLEY, Ph.D. Washington U., Dept. of Zool., St. Louis 5, Mo. *Assoc. Prof.* (2, 1954)

COHN, CLARENCE, M.D. Michael Reese Hosp., Med. Research Inst., Chicago, Ill. *Dir. of Dept. of Biochem.* (1, 1948)

COHN, MELVIN, Ph.D. Washington U. Sch. of Med., Dept. of Microbiol., St. Louis 10, Mo. *Prof.* (2, 1955)

COHN, MILDRED, Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Research Assoc. Prof. Biol. Chem.* (2, 1949)

COHN, WALDO E., Ph.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Biochemist.* (2, 1944)

COLE, KENNETH S., Ph.D. Natl. Inst. of Neurological Diseases & Blindness, NIH, Bethesda 14, Md. *Chief, Lab. of Biophysics.* (1, 1934)

COLE, LEONARD JAY, M.S. U. S. Naval Radiol. Defense Lab., Exptl. Pathol. Br., San Francisco 24, Calif. *Branch Head.* (1, 1957)

COLE, ROGER DAVID, Ph.D. Rockefeller Inst. for Med. Res., New York City 21. *Research Assoc.* (2, 1958)

COLE, RUFUS, M.D., D.Sc. Mount Kisco, N. Y. *Member Emer., Rockefeller Inst. for Med. Research.* (4R, 1924; 6R, 1917)

COLE, VERSA V., Ph.D., M.D. Caro State Hosp. for Epileptics, Caro, Mich. *Pharmacologist.* (3, 1941)

COLLETT, MARY E., Ph.D. 34 Weston Rd., Wellesley 81, Mass. (1R, 1921)

COLLIER, CLARENCE R., M.D. Coll. of Med. Evangelists, Loma Linda, Calif. *Assoc. Prof. of Physiol.* (1, 1957)

COLLIER, H. BRUCE, Ph.D. U. of Alberta, Dept. of Biochem., Edmonton, Canada. *Prof. and Head of Dept.* (2, 1944)

COLLINGS, WILLIAM DOYNE, Ph.D. Michigan State U., Dept. of Physiol., East Lansing. *Prof.* (1, 1944)

COLLINS, RUSSELL J., A.M., M.D. 3 Orange St., St. John, New Brunswick, Canada. (3, 1915)

COLLIP, J. B., Ph.D., M.D. U. of Western Ontario, London, Canada. *Dean of Med.; Dir., Div. of Med. Res., Natl. Res. Council.* (1, 1920; 2, 1920)

COLOWICK, SIDNEY P., Ph.D. Johns Hopkins U., McCollum-Pratt Inst., Baltimore 18, Md. *Prof. of Biol.* (2, 1944)

COMAN, DALE R., M.D. U. of Pennsylvania Sch. of Med., Dept. of Pathol., Philadelphia 4. *Prof. & Chmn.* (4, 1939)

COMAR, CYRIL LEWIS, Ph.D. Cornell U., Ithaca, N. Y. *Dir., Lab. of Radiation Biol.* (2, 1956; 5, 1951)

COMBS, GERALD F., Ph.D. U. of Maryland, Dept. of Poultry Husbandry, College Park. *Prof. of Poultry Nutrition.* (5, 1954)

COMMONER, BARRY, Ph.D. Washington U., Dept. of Botany, St. Louis 5, Mo. *Prof. of Plant Physiol.* (2, 1956)

COMROE, JULIUS H., JR., M.D. U. of California Med. Ctr., San Francisco 22. *Dir., Cardiovascular Res. Inst.; Prof., Physiol.* (1, 1943; 3, 1939)

CONDLIFFE, PETER, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chemist.* (2, 1957)

CONGDON, CHARLES C., M.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Pathologist.* (4, 1953)

CONIGLIO, JOHN G., Ph.D. Vanderbilt U. Med. Sch., Nashville 5, Tenn. *Assoc. Prof. of Biochem.* (2, 1957)

CONKLIN, RUTH E., Ph.D. Vassar Coll., Poughkeepsie, N. Y. *Prof. of Physiol.* (1, 1940)

CONN, ERIC E., Ph.D. U. of California, Dept. of Agric. Biochem., Davis. *Asst. Prof. of Plant Biochem.* (2, 1952)

CONN, HADLEY L., JR., M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Asst. Prof. of Med.; Estab. Invest., Amer. Heart Assn.* (1, 1955)

CONSOLAZIO, WILLIAM V., B.S. Natl. Science Fndn., Washington 25, D. C. *Program Dir. for Molecular Biol.* (2, 1949)

CONSTANTINIDES, PARIS, M.D., Ph.D. U. of British Columbia Med. Sch., Dept. of Anatomy, Vancouver 8, Canada. *Prof.* (1, 1956)

CONZELMAN, GAYLORD M., JR., Ph.D. Christ Hosp. Inst. of Med. Research, Cincinnati 19, Ohio. *Research Assoc.* (3, 1957)

COOK, DONALD HUNTER, Ph.D. U. of Miami, Miami 55, Fla. *Prof. of Chem.* (2R, 1929)

COOK, DONALD L., Ph.D. G. D. Searle & Co., P. O. Box 5110, Chicago 80, Ill. *Pharmacologist.* (3, 1952)

COOK, LEONARD, Ph.D. Smith Kline & French Labs., Dept. of Pharmacol., Philadelphia 1, Pa. *Asst. Sec. Head.* (3, 1955)

COOK, SHERBURNE F., Ph.D. U. of California, Dept. of Physiol., Berkeley 4. *Prof.* (1, 1958)

COOKE, ROBERT A., A.M., M.D. 60 E. 58th St., New York City. *Dir. of Dept. of Allergy, Roosevelt Hosp.* (6, 1920)

COOLIDGE, THOMAS B., M.D., Ph.D. U. of Chicago,

Abbot Hall, Chicago 37, Ill. *Prof., Dept. of Biochem. and Zoller Clinic.* (2, 1942)

COON, JULIUS M., M.D., Ph.D. Jefferson Med. Coll., Dept. of Pharmacol., Philadelphia 7, Pa. *Prof. and Head of Dept.* (3, 1941)

COON, MINOR J., Ph.D. U. of Michigan Med. Sch., Ann Arbor. *Prof. of Biol. Chem.* (2, 1952)

COON, ROBERT W., M.D. U. of Vermont Coll. of Med., Burlington. *Chmn. and Prof. of Pathol.* (4, 1955)

COONS, ALBERT H., M.D. Harvard U. Med. Sch., Boston 15, Mass. *Visiting Prof. Bacteriol. & Immunol.* (6, 1950)

COONS, CALLIE MAE, Ph.D. USDA, Human Nutrition Research Div., Washington 25, D.C. *Dir.* (5, 1933)

COOPER, CECIL, Ph.D. Western Reserve U. Sch. of Med., Dept. of Biochem., Cleveland 6, Ohio. *Asst. Prof.* (2, 1958)

COOPER, MERLIN L., M.D. Children's Hosp. Research Fndn., Elland Ave. and Bethesda, Cincinnati 29, Ohio. *Fellow.* (6, 1949)

COOPERMAN, JACK M., Ph.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City 29. *Asst. Prof. of Pediatrics.* (5, 1952)

COPELAND, D. EUGENE, Ph.D. 8705 Susanna Lane, Chevy Chase 15, Md. *Exec. Secy., Cell Biol., Div. Research Grants, NIH.* (1, 1951)

COPENHAVER, JOHN H., Ph.D. Dartmouth Coll., Hanover, N.H. *Assoc. Prof. of Zool.* (2, 1957)

COPLEY, ALFRED LEWIN, M.D. Charing Cross Hosp., Med. Res. Labs., London W.C. 2, England. *Dir. Exptl. Vasc. Res.* (1, 1944)

COPP, DOUGLAS HAROLD, M.D., Ph.D. U. of British Columbia, Dept. of Physiol., Vancouver, Canada. *Prof. and Head of Dept.* (1, 1951)

CORCORAN, ARTHUR CURTIS, C.M., M.D. Cleveland Clinic Fndn., Cleveland 6, Ohio. *Asst. Dir., Research Div.* (1, 1940; 3, 1955)

CORDY, DONALD R., D.V.M., Ph.D. U. of California Sch. of Vet. Med., Davis. *Assoc. Prof. Vet. Pathol.; Assoc. Pathologist.* (4, 1954)

COREY, EDWARD LYMAN, Ph.D. U. of Virginia Sch. of Med., Charlottesville. *Prof. of Physiol.* (1, 1931)

CORI, CARL F., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Biochem.* (2, 1925)

CORLEY, RALPH CONNER, Ph.D. Purdue U., Dept. of Chem., Lafayette, Ind. *Prof. of Biochem.* (2, 1927)

CORNATZER, W. E., Ph.D., M.D. U. of North Dakota Med. Sch., Dept. of Biochem., Grand Forks. *Prof. and Head of Dept.* (2, 1950)

CORNMAN, IVOR, Ph.D. Hazleton Labs., Box 333, Falls Church, Va. *Chief, Cellular Physiol. & Microbiol. Dept.* (1, 1956)

CORPER, H. J., M.D., Ph.D. 1295 Clermont St., Denver 20, Colo. *Dir. Emer. Research, Natl. Jewish Hosp. & U. Colo.* (2R, 1912)

CORSON, SAMUEL A., Ph.D. U. of Arkansas Sch. of Med., Little Rock. *Assoc. Prof. of Physiol. and Pharmacol.* (1, 1943)

COSTA, ERMINIO, M.D. Galesburg Research Hosp., Galesburg, Ill. *Med. Research Assoc.* (3, 1958)

COSTA, PASQUALE J., Ph.D., M.D. Seton Hall Coll. of Med. & Dent., Dept. of Pharmacol., Jersey City 4, N.J. *Assoc. Prof.* (3, 1958)

COTTEN, MARION deVEAUX, Ph.D. Emory U. Sch. of Med., Dept. of Pharmacol., Atlanta 22, Ga. (3, 1953)

COTTS, GERHARD K., M.D. The Dominion Arms, Arlington 4, Va. (3, 1937)

CO TUI, FRANK W., M.D. Creedmoor Inst., Queens Village 27, N.Y. *Assoc. Research Scientist and Head, Dept. of Physiol.* (3, 1931)

COTZIAS, GEORGE C., M.D. Brookhaven Natl. Lab., Dept. of Med., Upton, N.Y. *Head, Physiol. Div.* (4, 1954)

COUCH, JAMES RUSSELL, Ph.D. Texas A & M Coll., College Station. *Prof., Depts. of Poultry Sci., Biochem. & Nutrition.* (2, 1953; 5, 1950)

COULSON, E. JACK, Ph.D. USDA, Agric. Research Serv., Washington 25, D.C. *Biochemist, EURDD.* (6, 1955)

COULSON, ROLAND A., Ph.D. Louisiana State U. Sch. of Med., Dept. of Biochem., New Orleans 12. *Prof.* (2, 1954)

COURNAND, ANDRE FREDERIC, M.D. Bellevue Hosp., Chest Serv., C.D. Bldg., New York City 16. *Prof. of Med., Columbia U.* (1, 1944)

COVINO, BENJAMIN GENE, Ph.D. Tufts U. Med. Sch., Dept. of Pharmacol., Boston 11, Mass. *Asst. Prof.* (1, 1957)

COWEN, DAVID, M.D. Columbia U. Coll. of P. & S., New York City 32. *Assoc. Prof. of Neuropathol.* (4, 1951)

COWGILL, GEORGE RAYMOND, Ph.D. Yale U., Nutrition Lab., New Haven 11, Conn. *Prof. Nutrition.* (1R, 1923; 2, 1922; 5, 1933)

COWGILL, ROBERT W., Ph.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Biochem.* (2, 1958)

COX, ALVIN J., JR., M.D. Stanford U. Sch. of Med., 2398 Sacramento St., San Francisco 15, Calif. *Prof. of Pathol.* (4, 1949)

COX, GERALD J., Ph.D. U. of Pittsburgh Sch. of Dent., Pittsburgh 13, Pa. *Prof. of Dental Research.* (2, 1930; 5, 1935)

COX, HERALD R., Sc.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Dir., Virus Research.* (6, 1946)

COX, WARREN M., JR., Ph.D. Mead Johnson & Co., Evansville 21, Ind. *Tech. Dir. of Labs.* (2, 1935; 5, 1945)

CRAIG, FRANCIS NORTHROP, Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Chief, Applied Physiol. Br.* (1, 1946)

CRAIG, JOHN M., M.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Pathol.* (4, 1955)

CRAIG, L. C., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1938)

CRAMPTON, CHARLES F., Ph.D. U. of Florida Coll. of Med., Dept. of Pathol., Gainesville. *Asst. Prof.* (2, 1955)

CRAMPTON, E. W., Ph.D., Macdonald Coll., Macdonald, Quebec, Canada. *Prof. and Chmn. Dept. of Nutrition.* (5, 1940)

CRANDALL, DANA I., Ph.D. U. of Cincinnati, Coll. of Med., Cincinnati 19, Ohio. *Assoc. Prof. of Biol. Chem.* (2, 1951)

CRANDALL, LATHAN A., JR., M.D., Ph.D. Miles

Labs., Inc., Elkhart, Ind. *Dir., Miles-Ames Research Labs.* (1, 1930)

CRANE, FREDERICK L., Ph.D. U. of Wisconsin, Inst. for Enzyme Research, Madison. *Asst. Prof.* (2, 1957)

CRANE, ROBERT K., Ph.D. Washington U. Med. Sch., Dept. of Biol. Chem., St. Louis 10, Mo. *Assoc. Prof. of Biochem.* (2, 1953)

CRANEFIELD, PAUL F., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Prof. of Physiol.* (1, 1956)

CRANSTON, ELIZABETH M., Ph.D. U. of Minnesota Med. Sch., Dept. of Pharmacol., Minneapolis 14. *Asst. Prof.* (3, 1946)

CRAVENS, W. W., Ph.D. McMillen Feed Mills, Decatur, Ind. *Dir. of Feed Research and Nutrition.* (5, 1947)

CRAVER, BRADFORD N., Ph.D., M.D. Squibb Inst. for Med. Research, New Brunswick, N. J. *Dir. of Pharmacol.* (3, 1946)

CRAWFORD, MADELEINE FIELD, Ph.D. 1653 Central Ave., Needham 92, Mass. *Pine Manor Jr. Coll., Wellesley.* (1, 1933)

CRESCITELLI, FREDERICK, Ph.D. U. of California, Dept. of Zool., Los Angeles 24. *Prof.* (1, 1946)

CRETCHER, LEONARD H., Ph.D. 144 N. Dithridge St., Pittsburgh 13, Pa. (2R, 1930)

CRIEP, LEO H., M.D. The Bigelow, Pittsburgh 19, Pa. *Assoc. Prof. of Med., U. of Pittsburgh Sch. of Med.* (6, 1951)

CRISLER, GEORGE R., Ph.D., M.D. The New England Bldg., Winter Park, Fla. (1, 1930)

CRISMON, JEFFERSON MARTINEAU, M.D. Stanford U., Dept. of Physiol., Stanford, Calif. *Prof. and Exec. of Dept.* (1, 1944)

CRITTENDEN, PHOEBE J., Ph.D. Goucher Coll., Dept. of Physiol. and Bacteriol., Towson 4, Md. *Prof. and Chmn. of Dept.* (1, 1937; 3, 1937)

CROMARTIE, WILLIAM J., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Assoc. Prof. of Bacteriol. and Med.* (4, 1951)

CRONHEIM, GEORG E., Ph.D. Riker Labs., Inc., Northridge, Calif. *Dir., Biol. Sci.* (3, 1951)

CRONKITE, EUGENE P., M.D. Brookhaven Natl. Lab., Upton, N.Y. *Head, Patbol. Div.* (1, 1954)

CROSLEY, ARCHER P., JR., M.D. Hosp. of U. of Pennsylvania, Philadelphia 4. *Assoc. Prof. of Med.* (3, 1958)

CROWLEY, LEONARD V., M.D. U. of Vermont Coll. of Med., Burlington. *Asst. Prof. of Patbol.* (4, 1958)

CRUMPTON, CHARLES W., M.D. U. of Wisconsin Med. Sch., Madison 6. *Assoc. Prof. of Med.; Dir., Cardiovasc. Research Lab.* (3, 1952)

CSAKY, TIHAMER Z., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Assoc. Prof. of Pharmacol.* (1, 1956; 3, 1952)

CSONKA, F. A., Ph.D. USDA, Human Nutrition Research Div., Washington 25, D.C. *Sr. Chemist.* (2, 1924)

CULLEN, STUART C., M.D. U. of California Med. Ctr., Dept. of Anesthesia, San Francisco 22. *Prof.; Chmn. of Dept.* (3, 1944)

CULLER, ELMER A., Ph.D. U. of Rochester, Rochester 20, N.Y. *Prof. of Psychol. and Dir. of Lab.* (1R, 1936)

CULLUMBINE, HARRY, M.D. U. of Toronto, Dept. of Pharmacol., Toronto 5, Canada. *Prof. and Head of Dept.* (3, 1957)

CUNHA, TONY JOSEPH, Ph.D. U. of Florida, Dept. of Animal Husbandry & Nutrition, Gainesville. *Head of Dept.* (5, 1950)

CUNNINGHAM, LEON W., Ph.D. Vanderbilt U. Sch. of Med., Dept. of Biochem., Nashville, Tenn. *Asst. Prof.* (2, 1958)

CUNNINGHAM, LEW S., M.D. Marquette U. Sch. of Med., Milwaukee 3, Wis. *Asst. Prof. of Anatomy.* (2, 1957)

CUNNINGHAM, RAYMOND W., Ph.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Tech. Dir.* (3, 1941)

CUNNINGHAM, ROBERT SYDNEY, M.D., Sc.D. 1102 W. 43rd St., Richmond 25, Va. (1R, 1923)

CURETON, THOMAS KIRK, JR., Ph.D. U. of Illinois Sch. of Phys. Ed., Urbana. *Prof.; Dir., Phys. Fitness Res. Lab.* (1, 1946)

CURNEN, EDWARD C., M.D. U. of North Carolina Sch. of Med., Dept. of Pediatrics, Chapel Hill. *Prof. and Chmn. of Dept.* (6, 1941)

CURRAN, GEORGE L., M.D. U. of Kansas Med. Ctr., Dept. of Med., Kansas City 12. *Assoc. Prof.* (2, 1956)

CURTIS, GEORGE MORRIS, Ph.D., M.D., Sc.D. 379 Westland Ave., Columbus, Ohio. *Prof. Emer. of Surgery, Ohio State U.* (4R, 1933)

CURTIS, HOWARD J., Ph.D. Brookhaven Natl. Lab., Upton, N.Y. *Sr. Biologist & Chmn., Biol. Dept.* (1, 1940)

CURTIS, JACK M., Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D.C. *Asst. Div. Chief.* (2, 1951)

CUSHING, JOHN E., JR., Ph.D. U. of California, Santa Barbara Coll., Goleta, Santa Barbara County. *Assoc. Prof. of Bacteriol.* (6, 1952)

CUTTING, WINDSOR C., M.D. Stanford U. Sch. of Med., Stanford, Calif. *Prof. of Exptl. Therap.* (3, 1939)

DA COSTA, ESTHER, Ph.D. Chicago Teachers Coll., 6800 Stewart, Chicago 21, Ill. *Physiologist.* (1, 1953; 5, 1951)

DAFT, FLOYD SHELTON, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Dir.* (2, 1949; 5, 1941)

DAGGS, RAY GILBERT, Ph.D. Amer. Physiological Soc., 9650 Wisconsin Ave., Washington 14, D.C. *Exec. Secy.-Treas.* (1, 1935; 5, 1933)

DAHL, LEWIS KITCHENER, M.D. Brookhaven Natl. Lab., Upton, N.Y. *Sr. Scientist and Head, Research Med. Serv.* (4, 1953)

DALE, SIR HENRY H., M.D., F.R.S. The Wellcome Trust, 52 Queen Anne St., London W. 1, England. (3H, 1926)

DALE, HOMER ELDON, D.V.M., Ph.D. U. of Missouri, Dept. of Veterinary Physiol., Columbia. *Prof.* (1, 1956)

DALLDORF, GILBERT, M.D. Natl. Fndn. for Infantile Paralysis, 301 E. 42nd St., New York City 17. *Assoc. Med. Dir.* (4, 1947; 6, 1952)

DALTON, ALBERT J., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Cytologist.* (4, 1942)

DAM, HENRIK, Ph.D. Polytechnic Inst., Dept. of Biochem. and Nutr., Copenhagen K, Denmark.

Prof. and Dir. of Dept. (2, 1944; 5, 1943)

D'AMOUR, FRED E., Ph.D. U. of Denver, Denver 10, Colo. *Chmn., Dept. of Zool.* (1, 1934)

D'AMOUR, MARIE C., Ph.D., M.D. Olive View Sanatorium, Olive View, Calif. (1, 1934)

DANDLIKER, WALTER B., Ph.D. U. of Miami Sch. of Med., Dept. of Biochem., Coral Gables 34, Fla. *Assoc. Prof.* (2, 1955)

DANES, B. SHANNON, Ph.D. Columbia U., Coll. of P. & S., New York City 32. (1, 1956)

D'ANGELO, SAVINO A., Ph.D. Jefferson Med. Coll., Dept. of Anatomy, Philadelphia 7, Pa. *Prof. of Histol. & Embryol.* (1, 1947)

DANIEL, EDWIN E., Ph.D. U. of British Columbia Sch. of Med., Dept. of Pharmacol., Vancouver, Canada. *Asst. Prof.* (3, 1958)

DANIEL, LOUISE J., Ph.D. Cornell U., Savage Hall, Ithaca, N.Y. *Prof. of Biochem. and Nutrition.* (2, 1949; 5, 1956)

DANIELS, AMY L., Ph.D. 6 Rowland Ct., Iowa City, Iowa. (2R, 1919; 5R, 1933)

DANIELS, JOAN B., S.B. Massachusetts Dept. of Public Health, Boston. *Sr. Bacteriologist; Teacher, Simmons Coll. & Harvard.* (6, 1957)

DANIELSON, IRVIN S., Ph.D. American Cyanamid Co., Lederle Labs., Pearl River, N.Y. *Mgr., Biol. Production.* (2, 1937)

DANOWSKI, T. S., M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Renziehausen Prof. of Research Med.* (1, 1947)

DARBY, THOMAS DILLARD, Ph.D. Med. Coll. of South Carolina, Dept. of Pharmacol., Charleston 16. *Assoc.* (3, 1958)

DARBY, WILLIAM J., M.D., Ph.D. Vanderbilt U. Sch. of Med., Nashville 5, Tenn. *Prof. of Biochem.; Dir., Div. of Nutrition.* (2, 1949; 5, 1945)

DARLING, ROBERT CROLY, M.D. 157 Glenwood Ave., Leonia, N.J. *Prof., Phys. Med. & Rehabil., Columbia U.* (1, 1944)

DARROW, CHESTER W., Ph.D. Inst. for Juvenile Res., Chicago 12, Ill. *Psychophysiologist; Assoc. Prof. Physiol., U. of Ill.* (1, 1937)

DARROW, DANIEL CADY, M.D. U. of Kansas Med. Ctr., Dept. of Pediatrics, Kansas City. *Prof.* (2, 1936)

DASLER, WALDEMAR, Ph.D. Chicago Med. Sch., Chicago 12, Ill. *Assoc. Prof. of Biochem.* (5, 1957)

DAUBERT, B. F., Ph.D. General Foods Corp., Research Ctr., Tarrytown, N.Y. *Assoc. Dir., Chem. Research.* (2, 1947)

DAVENPORT, FRED M., M.D., Sc.Med.D. U. of Michigan Sch. of Public Health, Ann Arbor. *Assoc. Prof. Epidemiol. and Med.* (6, 1950)

DAVENPORT, HORACE WILLARD, Ph.D. U. of Michigan, Dept. of Physiol., Ann Arbor. *Prof. and Head of Dept.* (1, 1942)

DAVENPORT, VIRGINIA D., Ph.D. 1918 Day St., Ann Arbor, Mich. (1, 1950)

DAVEY, BESSIE L., Ph.D. U. of Alabama, Sch. of Home Econ., University. *Head, Dept. of Foods & Nutrition.* (5, 1957)

DAVID, NORMAN AUSTIN, M.D. U. of Oregon Med. Sch., Portland 1. *Prof. of Pharmacol.* (3, 1934)

DAVIDOW, BERNARD, Ph.D. New Drug Inst., 130 E. 59 St., New York City 22. *Dir. of Pharmacol.* (3, 1953)

DAVIDSOHN, ISRAEL, M.D. Mt. Sinai Hosp., Dept. of Pathol., Chicago 8, Ill. *Prof. and Chmn. of Dept. Pathol., Chicago Med. Sch.* (4, 1939; 6, 1929)

DAVIDSON, CHARLES S., M.D. Boston City Hosp., Boston 18, Mass. *Assoc. Dir., 2nd & 4th Med. Serv.; Assoc. Prof. of Med., Harvard U.* (5, 1949)

DAVIDSON, JACK D., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Sr. Investigator.* (3, 1958)

DAVIES, DEAN F., Ph.D., M.D. American Cancer Soc., 521 W. 57th St., New York City 19. *Admin. for Lung Cancer Research.* (1, 1951)

DAVIES, PHILIP W., Ph.D. Johns Hopkins U., Baltimore 5, Md. *Assoc. Prof. of Physiol. and Biophysics.* (1, 1948)

DAVIES, ROBERT E., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Biochem.* (2, 1957)

DAVIS, A. K., Ph.D. U. S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Asst. Head, Biophysics Br., Biomed. Div.* (1, 1956)

DAVIS, BERNARD D., M.D. Harvard Med. Sch., Boston 15, Mass. *Prof. of Bacteriol.; Chmn. of Dept.* (2, 1953; 3, 1955)

DAVIS, DORLAND J., M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Assoc. Dir. in Charge of Research.* (6, 1952)

DAVIS, GEORGE KELSO, Ph.D. Agric. Expt. Sta., Nutrition Lab., Gainesville, Fla. *Prof. of Nutrition, U. of Florida.* (2, 1956; 5, 1944)

DAVIS, HALLOWELL, M.D. Central Inst. for the Deaf, St. Louis 10, Mo. *Dir. of Research.* (1, 1925)

DAVIS, HARRY A., M.D. Coll. of Med. Evangelists, Dept. of Surgery, Los Angeles 57, Calif. *Clin. Prof. of Surgery.* (4, 1944)

DAVIS, JAMES O., Ph.D., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Head, Sec. on Exptl. CV Disease.* (1, 1954)

DAVIS, JOHN EMERSON, Ph.D. U. of Texas Coll. of Pharmacy, Austin. *Prof. of Pharmacol.* (1, 1941; 3, 1941)

DAVIS, M. E., M.D. Chicago Lying-In Hosp., Chicago 37, Ill. *Prof. & Chmn., Dept. of Obstetrics, U. of Chicago.* (3, 1950)

DAVIS, NEIL C., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Clin. Sci., Pittsburgh 13, Pa. *Assoc. Prof.* (2, 1957)

DAVIS, OSCAR F., M.D., Ph.D. Loyola U., Stritch Sch. of Med., Dept. of Pharmacol., & Michael Reese Hosp., Chicago, Ill. *Res. Assoc.* (3, 1956)

DAVIS, RICHARD LaVERN, M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Patbologist.* (4, 1958)

DAVISON, CLARKE, Ph.D. George Washington U. Sch. of Med., Washington 5, D.C. *Assoc. Prof. of Pharmacol.* (3, 1957)

DAWSON, CHARLES R., Ph.D. Columbia U., New York City 27. *Prof. of Chem.* (2, 1946)

DAWSON, JAMES ROBERTSON, JR., M.D. U. of Minnesota Sch. of Med., Minneapolis 14. *Prof. of Pathol.* (4, 1940)

DAWSON, PERCY M., M.D. 107 E. Portola Ave., Los Altos, Calif. (1R, 1900)

DAY, HARRY G., Sc.D. Indiana U., Dept. of Chem., Bloomington. *Prof. and Chmn. of Dept.* (2, 1948;

5, 1940)

DAY, PAUL L., Ph.D. U. of Arkansas Sch. of Med., Little Rock. *Prof. of Biochem.; Asst. Dean of Grad. Sch.* (2, 1934; 5, 1933)

DE, N. N., M.B. Central Drug Research Inst., Chattar Manjil Palace, Lucknow, U. P., India. *Asst. Dir. of Pharmacol.* (3, 1948)

DEARBORN, EARL H., Ph.D., M.D. American Cyanamid Co., Research Div., Pearl River, N. Y. *Head, Dept. Pharmacol. Research.* (3, 1946)

DEATHERAGE, F. E., Ph.D. Ohio State U., Dept. of Agric. Biochem., Columbus 10. *Prof. and Dept. Chmn.* (2, 1956)

DE BEER, EDWIN J., Ph.D. The Wellcome Research Labs., Tuckahoe, N. Y. *Assoc. Dir. of Research; Head, Dept. of Pharmacol.* (3, 1944)

DE BODO, RICHARD C., M.D. New York U. Coll. of Med., New York City 16. *Prof. of Pharmacol.* (1, 1932; 3, 1931)

DE BOER, BENJAMIN, Ph.D. U. of North Dakota Sch. of Med., Grand Forks. *Prof. of Physiol. and Pharmacol.* (1, 1947; 3, 1948)

DeBOER, CARL J., Ph.D. USDA Plum Is. Animal Disease Lab., Greenport, N. Y. *Sr. Scientist (Virol.).* (6, 1951)

DeEDS, FLOYD, Ph.D. 344 Santa Ana Ave., San Francisco, Calif. *Pharmacologist, USDA, WRRUD, Albany.* (2, 1937; 3, 1927)

DE GARA, P. F., M.D. 133 E. 58th St., New York City 22. *Assoc. Prof. Clin. Pediatrics, Cornell U. Med. Coll.; Physician, N. Y. Hosp.* (6, 1941)

DeGRAFF, ARTHUR C., M.D. New York Coll. of Med., New York City 16. *Prof. of Therapeutics.* (1, 1950; 3, 1937)

DE GUTIERREZ-MAHONEY, C. G., M.D. St. Vincent's Hosp., New York City 11. *Dir., Neurol. Div.; Neurosurgeon-in-Chief.* (1, 1940; 4, 1941)

DEICHMANN, WILLIAM B., Ph.D. U. of Miami Sch. of Med., Dept. of Pharmacol., Coral Gables 34, Fla. *Prof. and Chmn. of Dept.* (3, 1941)

DEKKER, CHARLES A., Ph.D. U. of California, Dept. of Biochem., Berkeley 4. *Assoc. Prof.* (2, 1956)

DE LA HUERGA, JESUS, M.D. Northwestern U., Chicago 12, Ill. *Asst. in Pathol.; Research Assoc., Hektoen Inst.* (4, 1956)

DELGADO, JOSE M. R., M.D. Yale U. Sch. of Med., New Haven, Conn. *Assoc. Prof. of Physiol. & Psychiatry.* (1, 1952)

DEL GRECO, FRANCESCO, M.D. Passavant Mem. Hosp., Chicago 11, Ill. (1, 1957)

DELORY, GEORGE EDWARD, Ph.D. U. of Manitoba Med. Coll., Biochem. Dept., Winnipeg, Canada. *Assoc. Prof.* (2R, 1951)

DEL POZO, E. C., M.D. Francisco Sosa #151, Coyoacan, D. F. 21, Mexico. *Prof. of Physiol., Univ. Nacional Autonoma.* (1, 1943)

DELWICHE, C. C., Ph.D. U. of California, Kearney Fndn., Berkeley 4. *Assoc. Prof.* (2, 1955)

DELWICHE, E. A., Ph.D. Cornell U. Coll. of Agric., Ithaca, N. Y. *Prof. of Bacteriol.* (2, 1956)

DeMAAR, EDMUND W. J., M.D., Ph.D. Merck Sharp & Dohme, West Point, Pa. (3, 1958)

DE MEIO, ROMANO HUMBERTO, Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Assoc. Prof. of Biochem.* (2, 1951)

DeMOSS, RALPH D., Ph.D. U. of Illinois, Dept. of Bacteriol., Urbana. *Assoc. Prof.* (2, 1956)

DEMPSEY, EDWARD W., Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Anatomy.* (1, 1940)

DEMPSHER, JOHN, M.D. U. of Pennsylvania Sch. of Med., Dept. of Pharmacol., Philadelphia 4. *Asst. Prof.* (3, 1958)

DENISON, ADAM B., JR., M.D. Bowman Gray Sch. of Med., Dept. of Physiol., Winston-Salem, N. C. *Asst. Prof.* (1, 1954)

DENNIS, EMERY W., Ph.D. Sterling-Winthrop Research Inst., Rensselaer, N. Y. *Dir., Biol. Div.* (3, 1952; 6, 1952)

DENNY, FLOYD W., JR., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Asst. Prof. Prev. Med. & Pediatrics.* (6, 1957)

DENSLOW, J. S., D.O. Kirksville Coll. of Osteopathy and Surgery, Still Mem. Res. Trust, Kirksville, Mo. *Prof.; Dir. Res. Affairs.* (1, 1949)

DENSTELT, ORVILLE F., Ph.D. McGill U., Dept. of Biochem., Montreal, Canada. *Assoc. Prof.* (2, 1948)

DENTON, CHARLES A., B.S. USDA, Animal and Poultry Husbandry Research Br., Beltsville, Md. *Chemist.* (5, 1954)

DERBYSHIRE, ARTHUR J., Ph.D. Harper Hosp., Detroit, Mich. *Head, EEG Dept.; Chmn. Res. Planning Team, Mich. Epilepsy Ctr.* (1, 1939)

DE RENZO, EDWARD C., Ph.D. Amer. Cyanamid Co., Research Div., Pearl River, N. Y. *Sr. Research Chemist, Group Leader.* (2, 1958)

DE SAVITSCH, EUGENE, M.D. 9, More's Garden, Cheyne Walk, London S. W. 3, England. (4, 1934)

DesMARAIS, ANDRE, Ph.D. U. of Ottawa, Dept. of Biol., Ottawa, Canada. *Prof.* (1, 1955)

DETHIER, VINCENT G., Ph.D. U. of Pennsylvania, Zool. Lab., Philadelphia 4. *Prof.* (1, 1950)

DETTWILER, HERMAN A., Ph.D. Eli Lilly & Co., Indianapolis, Ind. *Dir., Biol. Production Div.* (6, 1946)

DeTURK, WILLIAM E., Ph.D., M.D. Duke Hosp., Box 3105, Durham, N. C. *Assoc. Prof. of Pharmacol., Duke U.* (3, 1953)

DETWEILER, DAVID K., V.M.D. U. of Pa. Sch. of Veterinary Med., Philadelphia 4. *Assoc. Prof. of Pharmacol.; Chmn., Vet. Med. Sci., Grad. Sch.* (1, 1958)

DEULOFEU, VENANCIO, D. Chem. Casilla Correo 2539, Buenos Aires, Argentina. *Prof. of Organic Chem., U. of Buenos Aires.* (2, 1942)

DEUTSCH, HAROLD F., Ph.D. U. of Wisconsin, Dept. of Physiol. Chem., Madison 6. *Prof.* (2, 1948)

DEWEY, VIRGINIA C., Ph.D. Amherst Coll., Biol. Lab., Amherst, Mass. *Research Assoc., Dept. of Biol.* (2, 1949)

DEWS, PETER B., M.B., Ch.B., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Pharmacol.* (3, 1953)

DEXTER, LEWIS, M.D. Peter Bent Brigham Hosp., Boston, Mass. *Asst. Prof. of Med., Harvard Med. Sch.* (1, 1951)

DEY, FREDERICK L., Ph.D., M.D., 15 Smith Ave., Niantic, Conn. (1, 1945)

DEYRUP, INGRITH J., Ph.D. Barnard Coll., Columbia U., Dept. of Zool., New York City 27. *As-*

soc. Prof. (1, 1949)

DICKISON, H. L., Ph.D. Bristol Labs., Inc., Bldg. 6, Syracuse 1, N.Y. *Dir., Pharmacol. Research; Asst. Dir. Research.* (3, 1946)

DICKMAN, SHERMAN R., Ph.D. U. of Utah, 412 Med. Bldg., Salt Lake City. *Assoc. Prof. of Biochem.* (2, 1951)

DIENES, LOUIS, M.D. Massachusetts Gen. Hosp., Boston 14. *Consultant Bacteriologist.* (6, 1924)

DIERMEIER, HAROLD F., Ph.D. State U. of New York Med. Coll., Syracuse 10. *Asst. Prof. of Pharmacol.* (3, 1953)

DIETRICK, LaROY S., Ph.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Assoc. Prof. of Biochem.* (2, 1955)

DILL, DAVID BRUCE, Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Dept. Dir., Med. Research.* (1, 1941; 2, 1927)

DILLE, JAMES M., Ph.D., M.D. U. of Washington Sch. of Med., Dept. of Pharmacol., Seattle 5. *Prof. and Exec. Officer.* (3, 1939)

DILLON, JOHN B., M.D. U. of California, Sch. of Med., Los Angeles 24. *Prof. of Anesthesiol.* (3, 1958)

DILLON, ROBERT T., Ph.D. G. D. Searle & Co., Box 5110, Chicago 80, Ill. *Head, Analytical Div.* (2, 1934)

DI LUZIO, NICHOLAS R., Ph.D. U. of Tennessee Med. Units, Div. of Physiol., Memphis 3. *Asst. Prof.* (1, 1956)

DINGLE, JOHN H., Sc.D., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Prof. of Prev. Med.* (6, 1941)

DINNING, JAMES S., Ph.D. U. of Arkansas Sch. of Med., Dept. of Biochem., Little Rock. *Assoc. Prof.* (5, 1953)

DI PALMA, JOSEPH R., M.D. Hahnemann Med. Coll., Dept. of Pharmacol., Philadelphia 2, Pa. *Prof. & Head of Dept.* (1, 1943; 3, 1951)

DiSALVO, N. A., D.D.S., Ph.D. Columbia U. Sch. of Dent. & Oral Surg., New York City 32. *Prof. & Dir., Div. Orthodontics.* (1, 1957)

DISCHE, ZACHARIAS, M.D. Columbia U. Coll. of P. & S., Dept. of Ophthalmol., New York City 32. *Prof. of Biochem.* (2, 1944)

DiSTEFANO, VICTOR F., Ph.D. U. of Rochester Sch. of Med. & Dent., Rochester 20, N.Y. *Asst. Prof.* (3, 1958)

DITTMER, KARL, Ph.D. Florida State U., Dept. of Chem., Tallahassee. *Head of Dept.* (2, 1950)

DIXON, FRANK JAMES, JR., M.D. U. of Pittsburgh Sch. of Med., Dept. of Pathol., Pittsburgh 13, Pa. *Prof.* (4, 1951; 6, 1950)

DIXON, HAROLD M., M.D. VA, Veterans Benefits Office, Med. Div., 21st at Constitution Ave., Washington 25, D.C. *Asst. Chief Med. Officer.* (4, 1936)

DOAN, CHARLES A., M.D. Ohio State U. Coll. of Med., Columbus. *Dean; Dir., Med. Research; Prof. of Med.* (4, 1928)

DOBSON, ERNEST L., Ph.D. 15 Canyon Rd., Berkeley 4, Calif. *Physiologist, Radiation Lab.; Lect. in Physiol., U. of California.* (1, 1954)

DOCHEZ, A. RAYMOND, M.D. 1 W. 54 St., New York City 19. (4R, 1917; 6R, 1922)

DOCTOR, V. M., Ph.D. Texas Med. Ctr., Houston 25. *Assoc. Biochemist, U. of Texas, M. D. Anderson Hosp.* (2, 1957)

DODD, MATTHEW C., Ph.D. Ohio State U., Dept. of Bacteriol., Columbus 10. *Prof. of Bacteriol. and Immunol.* (6, 1950)

DODDS, MARY L., Ph.D. Pennsylvania State U., Coll. of Home Econ., University Park. *Prof., Foods & Nutrition.* (5, 1948)

DOHAN, F. CURTIS, M.D. 80 Princeton Rd., Bala-Cynwyd, Pa. *Asst. Prof. of Clin. Med., U. of Pennsylvania.* (1, 1941)

DOHERTY, DAVID G., Ph.D. Oak Ridge Natl. Lab., Biology Div., Oak Ridge, Tenn. *Sr. Biochemist.* (2, 1953)

DOISY, EDWARD A., SR., Ph.D. St. Louis U. Sch. of Med., St. Louis 4, Mo. *Prof. of Biol. Chem. and Dir. of Dept.* (2, 1920)

DOISY, EDWARD A., JR., M.D. St. Louis U. Sch. of Med., Dept. of Internal Med., St. Louis 4, Mo. *Assoc. Prof.* (2, 1951)

DOLE, VINCENT P., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (1, 1955)

DOLIN, M. I., Ph.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Biochemist.* (2, 1957)

DOLMAN, C. E., M.B., D.P.H., Ph.D. U. of British Columbia, Vancouver, Canada. *Head, Dept. of Bacteriol. & Immunol.* (6, 1947)

DOMINGUEZ, RAFAEL, M.D. St. Luke's Hosp., Cleveland 4, Ohio. *Head, Dept. Research, Div. Pathol.* (1, 1935)

DOMINO, EDWARD F., M.D. U. of Michigan Med. Sch., Dept. of Pharmacol., Ann Arbor. *Assoc. Prof.* (3, 1953)

DONAHUE, D. D., D.Sc. 4228 Military Rd., N.W., Washington 15, D.C. (3, 1941)

DONALDSON, DAVID M., Ph.D. Brigham Young U., Dept. of Bacteriol., Provo, Utah. *Assoc. Prof.* (6, 1956)

DONTAS, ANASTASIUS S., M.D. Evangelismos Hosp., Athens, Greece. *Assoc.* (3, 1956)

DOOLEY, MARION S., M.D. State U. of New York Med. Ctr., Syracuse. *Prof. of Pharmacol. Emer.; Hon. Member Staff, U. Hosp. Good Shepherd.* (3R, 1923)

DORFMAN, ALBERT, Ph.D., M.D. U. of Chicago, Dept. of Pediatrics, Chicago 11, Ill. *Prof.; Dir., La Rabida-U. of Chicago Inst.* (2, 1948)

DORFMAN, RALPH I., Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Dir. of Labs.; Prof. Biochem., Boston U.; Prof., Chem., Clark U.* (2, 1940)

DOTTI, LOUIS BASIL, Ph.D. St. Luke's Hosp., Amsterdam Ave. and 113th St., New York City 25. *Asst. Dir. of Labs.* (1, 1937)

DOTY, J. ROY, Ph.D. American Dental Assn., 222 E. Superior St., Chicago, Ill. *Secy., Council on Dental Therap.* (2, 1941)

DOTY, PAUL M., Ph.D. Harvard U., Dept. of Chem., Cambridge 38, Mass. *Prof.* (2, 1957)

DOTY, ROBERT W., Ph.D. U. of Michigan, Dept. of Physiol., Ann Arbor. *Assoc. Prof.* (1, 1951)

DOUDOROFF, MICHAEL, Ph.D. U. of California, Dept. of Bacteriol., 3517 Life Sciences Bldg., Berkeley 4. *Prof.* (2, 1946)

DOUGHERTY, ROBERT W., D.V.M. New York State Veterinary Coll., Cornell U., Ithaca. *Prof. of Veterinary Physiol.* (1, 1951)

DOUGHERTY, THOMAS F., Ph.D. U. of Utah Sch. of

Med., Dept. of Anatomy, Salt Lake City. *Prof. and Chmn. of Dept.* (1, 1950)

DOUGLAS, WILLIAM W., M.D. Albert Einstein Coll. of Med., Dept. of Pharmacol., New York City 61. *Assoc. Prof.* (1, 1957; 3, 1956)

DOULL, JOHN, Ph.D., M.D. USAF Radiation Lab., U. of Chicago, Chicago 37, Ill. *Asst. Dir.; Asst. Prof., Dept. of Pharmacol.* (3, 1953)

DOUNCE, ALEXANDER L., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Assoc. Prof. of Biochem.* (2, 1944)

DOUPE, JOSEPH, M.D. U. of Manitoba, Dept. of Physiol., Winnipeg, Canada. *Prof. and Head of Dept.* (1, 1949)

DOW, PHILIP, Ph.D. Med Coll. of Georgia, Augusta. *Prof. of Physiol.* (1, 1939)

DOW, ROBERT S., M.D., Ph.D. U. of Oregon Med. Sch., Portland 1. *Assoc. Clin. Prof. of Med. (Neurol.).* (1, 1940)

DOWBEN, ROBERT MORRIS, M.D. Northwestern U., Dept. of Med., Chicago 11, Ill. (1, 1957)

DOWNS, ARDREY W., M.D., D.Sc. U. of Alberta, Edmonton, Canada. *Emer. Prof. of Physiol. and Pharmacol.* (1R, 1917)

DOWNS, CORNELIA M., Ph.D. U. of Kansas, Dept. of Bacteriol., Lawrence. *Prof.* (6, 1929)

DOYLE, WILLIAM LEWIS, Ph.D. U. of Chicago, Dept. of Anatomy, Chicago 37, Ill. *Prof.* (1, 1946)

DRABKIN, DAVID L., M.D. U. of Pennsylvania Grad. Sch. of Med., Philadelphia 4. *Prof. and Chmn., Dept. of Biochem.* (2, 1928; 5, 1934)

DRAGSTEDT, CARL A., Ph.D., M.D. Northwestern U. Med. Sch., Dept. of Pharmacol., Chicago 11, Ill. *Prof.* (1, 1928; 3, 1932)

DRAGSTEDT, LESTER R., M.D., Ph.D. U. of Chicago, Dept. of Surgery, Chicago 37, Ill. *Prof.* (1, 1920)

DRAIZE, J. H., Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D.C. *Chief, Skin Toxicity Br.* (3, 1940)

DRAKE, T. G. H., M.B. 88 Admiral Rd., Toronto 5, Canada. (5, 1936)

DRAPER, WILLIAM B., M.Sc., M.D. U. of Colorado Sch. of Med., Denver 7. *Prof. of Pharmacol.* (3, 1938)

DREISBACH, ROBERT H., Ph.D., M.D. Stanford U. Sch. of Med., Dept. of Pharmacol., Stanford, Calif. *Prof.* (3, 1945)

DRELL, WILLIAM, Ph.D. U. of California Med. Ctr., Dept. Physiol. Chem., Los Angeles 24. *Estab. Invest., Amer. Heart Assn.* (2, 1955)

DRESEL, PETER E., Ph.D. U. of Manitoba, Faculty of Med., Dept. of Pharmacol. & Therap., Winnipeg, Canada. *Asst. Prof.* (3, 1955)

DREW, RUTH, Ph.D. Brookhaven Natl. Lab., Upton, N.Y. *Assoc. Med. Bacteriologist.* (6, 1957)

DREYER, NICHOLAS BERNARD. P.O. Box 72, Adelaide, Cape Province, Union of So. Africa. (3, 1942)

DRILL, VICTOR A., Ph.D., M.D. G. D. Searle & Co., Chicago 80, Ill. *Dir., Biol. Res.; Lect., Northwestern U. & U. of Ill.* (1, 1943; 3, 1946)

DRIPPS, ROBERT D., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Anesthesiol.* (1, 1947; 3, 1945)

DRIVER, ROBERT L., Ph.D., M.D. 3054 Franklin St., San Francisco, Calif. (1, 1945; 3, 1947)

DRURY, DOUGLAS R., M.D. U. of Southern California Sch. of Med., Dept. of Physiol., Los Angeles. *Prof. and Chmn. of Dept.* (1, 1932)

DRYDEN, LESLIE P., B.S. USDA, Agric. Research Serv., Dairy Cattle Research Br., Beltsville, Md. *Chemist.* (5, 1957)

DUBIN, HARRY E., Ph.D. 43 Calton Rd., New Rochelle, N.Y. (2, 1925)

DUBIN, ISADORE N., M.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. (4, 1947)

DUBNOFF, JACOB W., Ph.D. 1201 E. California St., Pasadena 4, Calif. *Sr. Research Fellow, California Inst. of Tech.* (2, 1946)

DU BOIS, ARTHUR B., M.D. U. of Pennsylvania Grad. Sch. of Med., Philadelphia 4. *Assoc. Prof. of Physiol.* (1, 1954)

DuBOIS, EUGENE F., M.D. 200 East End Ave., New York City 28. *Emer. Prof. of Physiol., Cornell U.* (1R, 1913; 3R, 1921; 5R, 1933)

DU BOIS, KENNETH P., Ph.D. U. of Chicago, Dept. of Pharmacol., Chicago 37, Ill. *Prof.; Dir., Air Force Radiation Lab.* (3, 1946)

DUBOS, RENE J., Ph.D., D.Sc. Rockefeller Inst. for Med. Research, New York City 21. *Member & Prof.* (6, 1938)

DUFFY, CARL E., Ph.D. U. of Arkansas Sch. of Med., Little Rock. *Prof. of Microbiol.* (6, 1951)

DUGAL, L. PAUL, Ph.D. U. of Ottawa Faculty of Sci., Dept. of Biol., Ottawa, Canada. *Dir. and Prof. of Biol.* (1, 1947)

DUGGAN, EDWARD L., Ph.D. U. of California Sch. of Med., 960 Med. Sci. Bldg., San Francisco 22. *Assoc. Prof. of Biochem.* (2, 1951)

DUKES, H. H., D.V.M. Cornell U., New York State Veterinary Coll., Ithaca. *Prof. of Veterinary Physiol.* (1, 1934)

DULANEY, ANNA D., Ph.D. U. of Tennessee Inst. of Pathol., Memphis. *Assoc. Prof. of Bacteriol., Med. Sch.* (6, 1924)

DUMKE, PAUL RUDOLPH, M.D. Henry Ford Hosp., Detroit 2, Mich. *Dir., Dept. of Anesthesiol.* (3, 1942)

DUMM, MARY E., Ph.D. New York U. Coll. of Med., New York City 16. *Asst. Prof. of Med.* (1, 1950)

DUN, FWU TARNG, Ph.D. Kirksville Coll. of Osteopathy & Surgery, Dept. of Physiol., Kirksville, Mo. *Prof.* (1, 1955)

DUNHAM, WOLCOTT B., M.D. Kennedy Hosp., VA Med. Teaching Group, Memphis 15, Tenn. *Dir., Med. Research Lab.* (6, 1951)

DUNLAP, CHARLES E., M.D. Tulane U. Sch. of Med., New Orleans 18, La. *Prof. of Pathol.* (4, 1942)

DUNN, ARTHUR L., Ph.D. VA Hosp., Radioisotope Serv., Omaha 5, Nebr. *Prin. Scientist.* (1, 1951)

DUNN, FLOYD W., Ph.D. Abilene Christian Coll., Abilene, Tex. *Prof. of Chem.* (2, 1957)

DUNN, MAX SHAW, Ph.D. U. of California, Los Angeles 24. *Prof. of Chem.; Prof. of Physiol. Chem.* (2, 1933)

DUNN, THELMA BRUMFIELD, M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Med. Officer.* (4, 1945)

DURBIN, RICHARD P., Ph.D. Harvard Med. Sch.,

Biophysical Lab., Boston 15, Mass. *Assoc. in Physiol.* (1, 1958)

DURRUM, EMMETT L., M.D. Stanford U., Dept. of Med. Microbiol., Stanford, Calif. *Assoc. Clin. Prof.* (2, 1954)

DURY, ABRAHAM, Ph.D. Bradford Hosp., Interstate Pkwy., Bradford, Pa. *Dir., Dorn Lab. for Med. Research.* (1, 1948)

DURYEE, WILLIAM R., Ph.D. George Washington U. Sch. of Med., Dept. of Physiol., Washington 5, D. C. *Research Prof.* (1, 1952)

DUTCHER, JAMES D., Ph.D. Squibb Inst. for Med. Research, New Brunswick, N. J. *Dept. Head, Div. of Organic Chem.* (2, 1946)

DUTCHER, R. A., D.Sc. 254 E. Hamilton Ave., State College, Pa. *Prof. Emer., Dept. Agric. & Biol. Chem., Pa. State U.* (2R, 1920; 5R, 1933)

DUVAL, CHARLES WARREN, M.D. 377 Pine St., South Weymouth, Mass. (4R, 1913)

DU VIGNEAUD, VINCENT, Ph.D. Cornell U. Med. Coll., New York City 21. *Prof. of Biochem.* (2, 1929; 5, 1934)

DWORETZKY, MURRAY, M.D. Cornell U. Med. Coll., New York City 21. *Asst. Prof. Clin. Med.; Asst. Prof. Clin. Public Health & Prev. Med.* (6, 1958)

DWORKIN, SIMON, D.D.S., M.D., M.Sc. McGill U., Biol. Bldg., Montreal, Canada. *Asst. Prof. in Physiol., Faculty of Med.* (1, 1931)

DYE, JOSEPH A., Ph.D. Cornell U., Ithaca, N. Y. *Prof. of Physiol.* (1, 1929)

DYE, MARIE, Ph.D. Michigan State U., East Lansing. *Dean (Emer.), Coll. of Home Econ.* (2R, 1929; 5R, 1933)

DYER, HELEN M., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1936; 5, 1937)

DZIEMIAN, ARTHUR J., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Dep. Chief, Biophysics Div.* (1, 1948)

DZIEWIATKOWSKI, DOMINIC D., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof., Biochem.* (2, 1950)

EADIE, GEORGE S., Ph.D. Duke U. Sch. of Med., Box 3709, Durham, N. C. *Prof. of Physiol. and Pharmacol.* (1, 1929; 3, 1940)

EAGLE, EDWARD, Ph.D. Swift and Co., Research Labs., Chicago 9, Ill. *Head, Div. of Physiol. and Toxicol.* (1, 1950; 3, 1953)

EAGLE, HARRY, M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Chief, Sec. Exptl. Therap.* (2, 1957; 3R, 1946; 6, 1953)

EAKIN, ROBERT E., Ph.D. U. of Texas, Dept. of Chem., Austin. *Prof.* (2, 1948)

EARLE, D. P., JR., M.D., Med. Sc.D. Northwestern U. Med. Sch., Chicago 11, Ill. (1, 1947)

EARLE, WILTON R., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Tissue Culture Sec., Lab. of Biol.* (4, 1940)

EASTON, DEXTER M., Ph.D. Florida State U., Tallahassee. *Assoc. Prof. of Physiol.* (1, 1955)

EATON, H. D., Ph.D. U. of Connecticut, Animal Industries Dept., Storrs. *Prof. of Animal Nutrition.* (5, 1957)

EATON, MONROE D., M.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. Prof. of Bacteriol. and Immunol.* (6, 1937)

ECCLES, JOHN CAREW, Ph.D. Australian Natl. U., Dept. of Physiol., Canberra, Australia. *Prof.* (1H, 1952)

ECKENHOFF, JAMES E., M.D. Hosp. of the U. of Pennsylvania, Philadelphia 4. *Prof. of Anesthesiol.* (1, 1948)

ECKER, E. E., Ph.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Prof. Emer. of Immunol.* (4, 1925; 6, 1947)

ECKERT, EDWARD A., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Prof. in Microbiol.* (4, 1954; 6, 1954)

ECKSTEIN, GUSTAV, M.D. U. of Cincinnati Coll. of Med., Cincinnati, Ohio. *Prof. of Physiol.* (1, 1948)

ECKSTEIN, HENRY C., Ph.D. U. of Michigan, 320 W. Med. Bldg., Ann Arbor. *Prof. of Biol. Chem.* (2, 1925)

ECKSTEIN, R. W., M.D. Western Reserve U., Cleveland, Ohio. *Asst. Prof. of Med. and Physiol.* (1, 1947)

EDDY, BERNICE E., Ph.D. Div. of Biologics Standards, NIH, Bethesda 14, Md. *Sr. Bacteriologist.* (6, 1949)

EDDY, NATHAN B., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Med. Officer.* (3, 1929)

EDDY, WALTER H., Ph.D. P. O. Box 6125, Southboro Sta., West Palm Beach, Fla. *Research Dir., Mangrove Products Co.* (2R, 1913; 5R, 1933)

EDELBERG, ROBERT, Ph.D. Baylor U. Coll. of Med., Dept. of Psychiatry and Neurol., Houston, Tex. *Research Assoc.* (1, 1956)

EDELHOCH, HAROLD, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. (2, 1957)

EDELMAN, ISIDORE S., M.D. U. of California Sch. of Med., San Francisco 22. *Assoc. Prof. of Med.* (1, 1955)

EDELMANN, ABRAHAM, Ph.D. Nuclear Science and Engineering Corp., Pittsburgh 36, Pa. (1, 1948)

EDER, HOWARD A., M.D. Albert Einstein Coll. of Med., New York City 61. *Assoc. Prof. of Med.* (2, 1956)

EDERSTROM, H. E., Ph.D. U. of North Dakota Sch. of Med., Dept. of Physiol., Grand Forks. *Prof.* (1, 1949)

EDHOLM, O. G., M.B. Natl. Inst. for Med. Research, Hampstead, London, N. W. 3, England. *Head, Div. of Human Physiol.* (1, 1948)

EDSALL, GEOFFREY, M.D. Walter Reed Army Inst. of Research, Washington 12, D. C. *Dir., Communicable Disease Div.* (6, 1943)

EDSALL, JOHN TILESTON, M.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Prof. of Biol. Chem.* (2, 1931)

EDWARDS, CHARLES, Ph.D. U. of Utah Coll. of Med., Dept. of Physiol., Salt Lake City 12. *Asst. Prof.* (1, 1956)

EDWARDS, DAYTON J., Ph.D. Cornell U. Med. Coll., New York City 21. *Secy. of the Faculty.* (1R, 1921)

EDWARDS, J. GRAHAM, Ph.D. 1410-20th St., N. W., Washington, D. C. (1, 1932)

EDWARDS, JESSE E., M.D. Mayo Clinic, Rochester 4, Minn. *Prof. of Pathol. Anatomy, Mayo Fndn.* (4, 1941)

EDWARDS, LESLIE E., Ph.D. Med. Coll. of Virgin-

ia, Dept. of Physiol., Richmond. *Assoc. Prof.* (1, 1950)

EGDAHL, RICHARD H., M.D., Ph.D. Med. Coll. of Virginia, Richmond 19. *Dir., Surgical Research Labs.* (4, 1957)

EGGERTH, ARNOLD H., Ph.D. 406 St. John's Pl., Brooklyn, N.Y. *Visiting Prof. Microbiol. & Immunol., Albert Einstein Med. Coll.* (4, 1925)

EHRENSTEIN, MAXIMILIAN R., Ph.D. U. of Pennsylvania Hosp., Maloney Clinic, Philadelphia 4. *Prof. of Biochem., U. of Pa.* (2, 1942)

EHRICH, W. E., M.D. U. of Pennsylvania, Grad. Sch. of Med., Philadelphia 4, Pa. *Prof. Pathol.; Chief, Div. Pathol., Philadelphia Gen. Hosp.* (4, 1945)

EICHEL, HERBERT J., Ph.D. Hahnemann Med. Coll., Div. of Biol. Chem., Philadelphia 2, Pa. *Asst. Prof.* (2, 1957)

EICHELBERGER, LILLIAN, Ph.D. U. of Chicago, Dept. of Surgery, Chicago 37, Ill. *Assoc. Prof. of Biochem.* (2, 1937)

EICHNA, LUDWIG W., M.D. New York U. Coll. of Med., New York City 16. *Prof., Med.; Visiting Physician, Bellevue Hosp.* (1, 1949)

EICHWALD, ERNEST J. Montana Deaconess Hosp., Great Falls. (4, 1953)

EIK-NES, KRISTEN B., M.D. U. of Utah Coll. of Med., Dept. of Biochem., Salt Lake City 12. *Asst. Res. Prof.; Training Program Dir.* (1, 1958)

EILER, JOHN J., Ph.D. U. of California Med. Ctr., San Francisco 22. *Prof. of Biochem. and Pharmaceut. Chem.* (2, 1948)

EISEN, HERMAN N., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Med. (Dermatol.).* (6, 1951)

EISENBERG, FRANK, JR., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Biochemist.* (2, 1955)

EISENBERG, MAX A., Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Asst. Prof.* (2, 1958)

EISENBRANDT, LESLIE L., Ph.D. U. of Kansas City, Kansas City 10, Mo. *Prof. of Pharmacol.; Dean, Sch. of Pharmacy.* (3, 1951)

EISENMAN, ANNA J., Ph.D. USPHS Hosp., NIMH Addiction Research Ctr., Lexington, Ky. *Biochemist.* (2, 1930)

EISENMAN, GEORGE, M.D. Eastern Pennsylvania Psychiatric Inst., Dept. of Basic Research, Philadelphia 29. *Sr. Staff Scientist.* (1, 1955)

EKLUND, CARL M., M.D. Rocky Mountain Lab., Hamilton, Mont. *Invest., Mosquito and Tick Transmitted Virus Diseases.* (4, 1957)

ELBERG, SANFORD S., Ph.D. U. of California, Dept. of Bacteriol., Berkeley 4. *Prof.* (6, 1948)

ELDERFIELD, ROBERT C., Ph.D., D.Sc. U. of Michigan, 2020 Chemistry Bldg., Ann Arbor. *Prof. of Chem.* (2, 1934)

ELDRED, EARL, M.D. U. of California, Dept. of Anatomy, Los Angeles 24. *Assoc. Prof.* (1, 1956)

ELFTMAN, HERBERT, Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Anatomy.* (1, 1940)

ELION, GERTRUDE B., M.S. 270 Bronxville Rd., Bronxville 8, N.Y. *Sr. Biochemist, Wellcome Research Labs.* (2, 1952)

ELIOT, MARTHA M., M.D. 21 Francis Ave., Cambridge 38, Mass. *Prof. of Maternal and Child Health, Harvard Sch. of Public Health.* (5, 1933)

ELKES, JOEL J., M.D. St. Elizabeths Hosp., Clin. Neuropharmacol. Res. Ctr., Washington 20, D.C. *Chief, Clin. Neuropharmacol.* (3, 1958)

ELKINTON, J. RUSSELL, M.D. U. of Pennsylvania Hosp., Dept. of Med., Philadelphia 4. (1, 1947)

ELLENBOGEN, ERIC, Ph.D. U. of Pittsburgh Grad. Sch. Public Health, Dept. Biochem. and Nutr., Pittsburgh 13, Pa. *Assoc. Prof.* (2, 1956)

ELLINGER, FRIEDRICH P., M.D. Naval Med. Research Inst., Bethesda 14, Md. *Chief Radiation Biologist; Head of Pharmacol. Div.* (3, 1955)

ELLIOTT, HENRY W., Ph.D., M.D. U. of California Med. Ctr., San Francisco 22. *Asst. Prof. of Pharmacol. and Anesthesia.* (3, 1948)

ELLIOTT, JOSEPH ROBERT, Ph.D. Jefferson Davis Hosp., 3rd Floor Lab., Houston, Tex. *Biochemist.* (1, 1957)

ELLIOTT, K. ALLAN C., Ph.D., Sc.D. Montreal Neurol. Inst., Montreal, Canada. *Assoc. Prof. of Exptl. Neurol. and Biochem.* (2, 1937)

ELLIOTT, WILLIAM H., Ph.D. St. Louis U. Sch. of Med., St. Louis 4, Mo. *Assoc. Prof. of Biochem.* (2, 1952)

ELLIS, C. H., Ph.D. Wellcome Research Labs., Tuckahoe 7, N.Y. *Sr. Pharmacologist.* (1, 1947)

ELLIS, FRED W., M.D., Ph.D. U. of North Carolina Sch. of Med., Chapel Hill. *Assoc. Prof. of Pharmacol.* (3, 1945)

ELLIS, JOHN T., M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Pathol. and of Pathol. in Surgery.* (4, 1956)

ELLIS, LILLIAN N., Ph.D. Rutgers U., Douglass Coll., New Brunswick, N.J. *Assoc. Prof. of Chem.* (5, 1940)

ELLIS, N. R., M.S. USDA, Agric. Research Ctr., Beltsville, Md. *Assoc. Dir., Animal Husbandry Res. Div.* (2, 1928; 5, 1934)

ELLIS, SYDNEY, Ph.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. *Prof. of Pharmacol. and Chmn. of Dept.* (3, 1947)

ELLISON, SOLON ARTHUR, D.D.S., Ph.D. Columbia U., Coll. of P. & S., Dept. of Microbiol., New York City 32. *Assoc.* (6, 1956)

ELMADJIAN, FRED, Ph.D. Box 57, Worcester, Mass. *Dir. of Labs., Worcester State Hosp.; Sr. Scientist, Worcester Fndn. for Exptl. Biol.* (1, 1950)

ELSON, DAVID, Ph.D. Weizmann Inst. of Science, Rehovot, Israel. *Sr. Scientist.* (2, 1956)

ELVEHJEM, CONRAD ARNOLD, Ph.D. U. of Wisconsin, Biochem. Bldg., Madison. *Prof.* (2, 1931; 5, 1933)

ELWELL, LEONARD H., Ph.D. U. of Michigan, Dept. of Physiol., Ann Arbor. *Asst. Prof.* (1, 1956)

ELWYN, DAVID, Ph.D. Harvard Med. Sch., Dept. of Biol. Chem., Boston 15, Mass. *Asst. Prof.* (2, 1958)

EMBREE, NORRIS DEAN, Ph.D. Distillation Products Industries, Rochester 3, N.Y. *Dir. of Tech. Operations.* (2, 1946)

EMERSON, GEORGE A., Ph.D. U. of Texas Med. Br., Galveston. *Prof. of Pharmacol.* (3, 1935)

EMERSON, GLADYS A., Ph.D. U. of California, Dept. of Home Econ., Los Angeles 24. *Chmn.;*

Prof. Home Econ. and Nutr. (5, 1942)

EMERSON, JACK DREW, Ph.D. U. of Alabama Med. Ctr., Dept. of Physiol., Birmingham 3. *Assoc. Prof.* (1, 1957)

EMMART, EMILY W., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Cytologist.* (3, 1947)

ENDERS, JOHN F., Ph.D. Children's Med. Ctr., Boston, Mass. *Chief, Research Div., Infectious Diseases; Prof., Harvard Med. Sch.* (6, 1936)

ENDICOTT, KENNETH M., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Cancer Chemotherapy Natl. Serv. Ctr., PHS.* (4, 1947)

ENGEL, FRANK L., M.D. Duke U. Sch. of Med., Durham, N. C. *Prof. of Med.; Assoc. Prof. of Physiol.* (1, 1947)

ENGEL, LEWIS L., Ph.D. Massachusetts Gen. Hosp. Boston 14. *Assoc. Prof. of Biol. Chem., Harvard Med. Sch.* (2, 1949)

ENGEL, RUBEN W., Ph.D. Virginia Polytechnic Inst., Dept. of Biochem. and Nutrition, Blacksburg. *Head of Dept.* (5, 1948)

ENGLARD, SASHA, Ph.D. Albert Einstein Coll. of Med., Dept. of Biochem., New York City 61 *Asst. Prof.* (2, 1958)

ENGLISH, JAMES, JR., Ph.D. Yale U., Sterling Chem. Lab., New Haven 11, Conn. *Assoc. Prof. of Chem.* (2, 1946)

ENRIGHT, JOHN B., Ph.D. U. of California Sch. of Veterinary Med., Davis. *Assoc. Prof.* (6, 1948)

ENTENMAN, CECIL, Ph.D. U. S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Head, Biochem. Br.* (1, 1953)

EPPRIGHT, ERCEL S., Ph.D. Iowa State Coll., Dept. of Food and Nutrition, Ames. *Prof. and Head of Dept.* (5, 1954)

EPSTEIN, ALBERT A., M.D. Stebbins Rd., Carmel, N. Y. (2R, 1912)

ERCOLI, N., M.D., Ph.D. Armour Labs., Kankakee, Ill. *Dir. of Pharmacol.* (3, 1947)

ERICKSON, CYRUS C., M.D. U. of Tennessee, Inst. of Pathol., Memphis 3. *Prof. of Pathol.* (4, 1941)

ERICKSON, JOHN OTTO, Ph.D. VA Ctr., Radioisotope Lab., Los Angeles 25, Calif. *Biophysicist.* (6, 1946)

ERLANGER, BERNARD F., Ph.D. Columbia U., Coll. of P. & S., Dept. of Microbiol., New York City 32. *Asst. Prof.* (2, 1956)

ERLANGER, JOSEPH, M.D., Sc.D. 5127 Waterman Blvd., St. Louis 8, Mo. *Prof. Emer. of Physiol.* (1R, 1901)

ERSHOFF, BENJAMIN H., Ph.D 9331 Venice Blvd., Culver City, Calif. *Dir., Western Biol. Labs.; Visiting Prof., U. Southern Calif.* (5, 1950)

ESCHENBRENNER, ALLEN B., M.D. PHS Communicable Disease Ctr., P. O. Box 185, Chamblee, Ga. (4, 1946)

ESPE, DWIGHT L., Ph.D. USDA State Expt. Stations Div., Agric. Research Serv., Washington 25, D. C. *Admin.* (1, 1940)

ESPLIN, DON W., Ph.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City. *Asst. Research Prof.* (3, 1957)

ESSEX, HIRAM E., Ph.D. Mayo Clinic, Rochester, Minn. *Emer. Prof. of Physiol., Grad. Sch., U. of Minn. under the Mayo Fndn.* (1, 1932; 3R, 1940)

ESSIG, CARL F., M.D. PHS Hosp., Lexington, Ky. *Sr. Surgeon, PHS.* (1, 1952)

ETSTEN, BENJAMIN E., M.D. New England Ctr. Hosp., Boston 11, Mass. *Anesthetist-in-Chief; Prof. of Anesthesia, Tufts U. Sch. of Med.* (3, 1954)

ETTINGER, G. H., M.D., C.M. Queen's U., Kingston, Canada. *Prof. of Physiol.; Dean, Faculty of Med.* (1, 1943)

EVANS, CHARLES A., M.D., Ph.D. U. of Washington, Dept. of Microbiol., Seattle 5. *Prof.* (6, 1952)

EVANS, E. EDWARD, Ph.D. U. of Alabama Med. Ctr., Dept. of Microbiol., Birmingham 3. *Assoc. Prof.* (6, 1955)

EVANS, EARL ALISON, JR., Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Prof. and Chmn. of Dept.* (2, 1939)

EVANS, F. GAYNOR, Ph.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Prof. of Anatomy.* (1, 1954)

EVANS, GERALD T., M.D., Ph.D. U. of Minnesota Hosps., Minneapolis. *Dir. of Clin. Labs.; Prof. of Med., U. of Minnesota.* (1, 1942)

EVANS, HERBERT M., M.D., D.Sc., LL.D. U. of California, Berkeley 4. *Prof. Emer. of Anatomy; Dir. Emer. of Inst. of Exptl. Biol.* (1R, 1919)

EVANS, JOHN DAVID, Ph.D. Temple U. Sch of Med., Philadelphia 40, Pa. *Assoc. Prof. of Physiol.* (1, 1952)

EVANS, ROBERT JOHN, Ph.D. Michigan State U., Dept. of Agric. Chem., East Lansing. *Research Prof.* (5, 1951)

EVANS, TITUS C., Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Prof. and Head of Radiation Research Lab.* (1, 1949)

EVANS, VIRGINIA J., Sc.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biologist (Cytol.).* (4, 1957)

EVARTS, EDWARD V., M.D. Natl. Inst. of Mental Health, Lab. of Clin. Sci., NIH, Bethesda 14, Md. *Chief, Sec. on Physiol.* (1, 1958)

EVELETH, D. F., Ph.D., D.V.M. North Dakota Agric. Coll., Fargo. *Prof. of Vet. Science, North Dakota Agric. Expt. Sta.* (2, 1939)

EVERETT, GUY M., Ph.D. Abbott Labs., Dept. of Pharmacol., North Chicago, Ill. *Group Leader, Neuropharmacol.* (3, 1950)

EVERETT, JOHN W., Ph.D. Duke Hosp., Box 3209, Durham, N. C. *Prof. of Anatomy, Duke U. Sch. of Med.* (1, 1951)

EVERETT, MARK R., Ph.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Dean; Dir., U. of Okla. Med. Ctr.* (2, 1929)

EVERETT, NEWTON B., Ph.D. U. of Washington Sch. of Med., Dept. of Anatomy, Seattle 5. *Prof. and Admin. Officer.* (1, 1956)

EVERSOLE, WILBURN J., M.Sc., Ph.D. U. of New Mexico, Dept. of Biol., Albuquerque. *Prof.* (1, 1950)

EVERSON, GLADYS, Ph.D. U. of California, Dept. of Home Econ., Davis. *Prof. and Chmn.* (5, 1948)

EYSTER, JOHN A. ENGLISH, M.D. Matlacha Sta., Ft. Myers, Fla. (1R, 1906; 3R, 1908)

FAHEY, JOHN L., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Sr. Invest., Metab. Serv.* (1, 1957)

FAHR, GEORGE, M.D. U. of Minnesota Med. Sch., Minneapolis. *Emer. Prof. of Int. Med.* (1R, 1913;

3R, 1940)

FAILEY, CRAWFORD F., Ph.D. 416 S. 6th St., Terre Haute, Ind. (2, 1933)

FAIRBAIRN, DONALD, Ph.D. Box 199, Macdonald Coll., Quebec, Canada. *Assoc. Prof. of Parasitol., McGill U.* (2, 1953)

FAIRLEY, JAMES L., Ph.D. Michigan State U., Kedzie Chem. Lab., East Lansing. *Assoc. Prof., Chem. Dept.* (2, 1955)

FALES, FRANK W., Ph.D. Emory U., Dept. of Biochem., Atlanta 22, Ga. *Asst. Prof.* (2, 1957)

FALK, CAROLYN R., B.A. 40 E. 66th St., New York City. *Sr. Bacteriologist (Retired) Bureau of Labs., Dept. of Health.* (6, 1943)

FALK, GERTRUDE, Ph.D. U. of Washington Sch. of Med., Dept. of Pharmacol., Seattle 5. *Asst. Prof.* (1, 1956; 3, 1957)

FAMULENER, LEMUEL W., Ph.C., M.D. 605 Main St., Owego, N.Y. (6R, 1920)

FARAH, ALFRED E., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Prof. of Pharmacol.* (3, 1948)

FARBER, EMMANUEL, M.D., Ph.D. Tulane U. Sch. of Med., Depts. Pathol. & Biochem., New Orleans 12, La. *Assoc. Prof.* (2, 1956; 4, 1953)

FARBER, SAUL J., M.D. New York U., Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (1, 1955)

FARBER, SIDNEY, M.D. 300 Longwood Ave., Boston 15, Mass. *Prof. of Pathol., Harvard Med. Sch. at Children's Hosp.* (4, 1934)

FARMER, CHESTER J., A.M. Wilson's Subdivision, Rt. 2, Cedar Lake, Ind. *Prof. Emer. of Biochem., Northwestern U.* (2R, 1935)

FARNER, DONALD S., Ph.D. State Coll. of Washington, Dept. of Zool., Pullman. *Prof. of Zoophysiol.* (1, 1952)

FARNSWORTH, EDITH B., M.D. 700 North Michigan, Chicago 11, Ill. *Asst. Prof. in Med., Northwestern U.* (3, 1952)

FARR, LEE E., M.D. Brookhaven Natl. Lab., Upton, N.Y. *Med. Dir.; Physician-in-Chief, Brookhaven Natl. Lab. Hosp.* (4, 1941)

FARR, RICHARD S., M.D. U. of Pittsburgh, Sch. of Med., Dept. of Med., Pittsburgh 13, Pa. *Asst. Prof.* (4, 1957; 6, 1955)

FARRAR, GEORGE E., JR., M.D. Wyeth Labs., Box 8299, Philadelphia 1, Pa. *Med. Dir.* (3, 1947)

FARRELL, GORDON L., M.D. Western Reserve U., Cleveland, Ohio. *Asst. Prof. of Physiol.* (1, 1956)

FARRELL, JAMES, Ph.D., M.D. 636 Church St., Evanston, Ill. (1, 1938)

FASSETT, DAVID W., M.D. Eastman Kodak Co., Lab. of Indust. Med., Kodak Park Works, Rochester, N.Y. *Dir. of Lab.* (3, 1942)

FAULCONER, ALBERT, JR., M.D. Mayo Clinic, Rochester, Minn. *Head, Sec. of Anesthesiol.* (3, 1950)

FAY, MARION, Ph.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. *Dean; Prof. of Physiol. Chem.* (2, 1937)

FAZEKAS, JOSEPH F., M.D. D.C. Gen. Hosp., Washington 3, D.C. *Chief of Staff.* (3, 1953)

FEATHERSTONE, ROBERT M., Ph.D. U. of California Sch. of Med., Dept. of Pharmacol., San Francisco 22. *Prof. and Chmn. of Dept.* (3, 1947)

FEDOR, EDWARD JOHN, Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Surgery, Pittsburgh 13, Pa. *Asst. Research Prof.* (1, 1958)

FEENEY, ROBERT EARL, Ph.D. U. of Nebraska Coll. of Agric., Dept. of Biochem. and Nutrition, Lincoln. *Prof. and Chmn. of Dept.* (2, 1952)

FEIGELSON, PHILIP, Ph.D. 99 Ft. Washington Ave., New York City 32. *Asst. Prof. of Biochem., Columbia U., Coll. of P. & S.* (2, 1957)

FEIGEN, GEORGE A., Ph.D. Stanford U., Dept. of Physiol., Stanford, Calif. *Assoc. Prof.* (1, 1948)

FEINBERG, RICHARD J., Ph.D., M.D. Research and Educational Hosp., 1422 N. Sedgwick St., Chicago 10, Ill. *Intern.* (6, 1958)

FEINBERG, SAMUEL M., M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Prof. of Med.; Chief, Allergy Clinic.* (6, 1955)

FEINGOLD, DAVID S., Ph.D. U. of California, Dept. of Agric. Biochem., Berkeley 4. *Jr. Asst. Research Biochemist.* (2, 1958)

FEINSTEIN, ROBERT N., Ph.D. Argonne Natl. Lab., Lemont, Ill. (2, 1950)

FELDMAN, HARRY A., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Prof. & Chmn., Dept. of Prev. Med.* (6, 1943)

FELDMAN, JOSEPH D., M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Prof. of Pathol.* (4, 1955)

FELDMAN, WILLIAM H., D.V.M., D.Sc. Veterans Admin., Dept. Med. & Surgery, Washington 25, D.C. *Chief, Lab. of Res. in Pulmonary Diseases.* (4, 1934)

FELLER, A. E., M.D. U. of Virginia Sch. of Med., Dept. of Microbiol., Charlottesville. *Prof. and Chmn. of Dept.* (6, 1943)

FELLER, DAVID D., Ph.D. VA Hosp., Seattle 8, Wash. *Res. Asst. Prof. of Med., U. of Washington; Prin. Scientist, Radioisotope Serv.* (1, 1951)

FELLOWS, EDWIN J., Ph.D. Smith Kline & French Labs., Philadelphia 1, Pa. *Assoc. Dir. of Research.* (3, 1939)

FELSENFELD, OSCAR, Walter Reed Army Inst. of Research, Washington 12, D.C. *Lt. Col. MC, USAR; Epidemiologist.* (6, 1949)

FELTS, JAMES M., Ph.D. Tufts U. Sch. of Med., Boston 11, Mass. *Asst. Prof. of Physiol.* (1, 1957)

FENN, WALLACE OSGOOD, Ph.D. U. of Rochester Med. Ctr., Rochester 20, N.Y. *Prof. of Physiol. and Chmn. of Dept.* (1, 1924)

FENNING, CON, M.D. U. of Utah Sch. of Med., Salt Lake City 1. *Prof. of Pharmacol. and Physiol.* (1, 1942)

FENTON, P. F., Ph.D. Brown U., Providence, R.I. *Prof. of Biol.* (1, 1947; 5, 1949)

FERGUSON, FRANK C., JR., M.D. Albany Med. Coll., Dept. of Pharmacol., Albany 8, N.Y. *Prof. and Chmn.* (3, 1951)

FERGUSON, FREDERICK P., Ph.D. U. of Maryland Sch. of Med., Dept. of Physiol., Baltimore 1. *Prof.* (1, 1949)

FERGUSON, J. K. W., M.D. 76 Kilbarry Rd., Toronto, Canada. *Prof. Pharmacol. and Dir., Connaught Med. Res. Labs., U. of Toronto.* (1, 1933; 3, 1941)

FERGUSON, JOHN, Ph.D. Creighton U. Sch. of Med., Omaha 2, Nebr. *Assoc. Prof. of Physiol. and*

Pharmacol. (1, 1949)

FERGUSON, J. H., M.D., D.Sc. U. of North Carolina Sch. of Med., Dept. of Physiol., Chapel Hill. *Prof. of Physiol. and Dept. Head.* (1, 1933)

FERGUSON, L. KRAEER, M.D. 133 S. 36th St., Philadelphia 4, Pa. *Prof. of Surgery, Grad. Scb., U. of Pa. & Woman's Med. Coll.; Surgeon.* (4, 1935)

FERGUSON, LLOYD C., D.V.M., Ph.D. Michigan State U., Dept. Microbiol. & Public Health, East Lansing. *Prof. & Head of Dept.* (6, 1953)

FERGUSON, RALPH L., M.D. 420 E. 33rd St., Joplin, Mo. *Pathologist, St. John's Hosp.* (4, 1949)

FERRIS, BENJAMIN G., JR., M.D. Town House Rd., Weston 93, Mass. *Assoc. Prof., Environ. Hygiene, Harvard Scb. Public Health.* (1, 1951)

FERRY, JOHN DOUGLASS, Ph.D. U. of Wisconsin, Madison. *Prof. of Chem.* (2, 1941)

FERRY, RONALD M., M.D. Barnes Hill Rd., Concord, Mass. *Master Emer. and Assoc. Prof. Emer. of Biochem., Harvard U.* (2, 1924)

FETCHER, EDWIN S., Ph.D. Steamboat Springs, Colo. (1, 1944)

FETTER, DOROTHY, Ph.D. Brooklyn Coll., Dept. of Home Econ., Brooklyn, N. Y. *Asst. Prof.* (1, 1944)

FEVOLD, HARRY L., Ph.D. Baxter Labs., Inc., 6301 Lincoln Ave., Morton Grove, Ill. *Research Coordinator.* (2, 1942)

FIELD, JOHN, II, Ph.D. U. of California Sch. of Med., Dept. of Physiol., Los Angeles 24. *Prof. and Chmn. of Dept.* (1, 1930)

FIELD, JOHN B., M.D., Ph.D. U. of Southern California Sch. of Med., Los Angeles 33. *Asst. Prof. of Clin. Med.* (2, 1952)

FILLEY, GILES F., M.D. U. of Colorado Med. Ctr., Denver 20. *Asst. Prof. of Med.* (1, 1957)

FINCH, CLEMENT A., M.D. 4516-47th Ave., N. E., Seattle 5, Wash. (1, 1951)

FINCKE, MARGARET L., Ph.D. Oregon State Coll., Sch. of Home Econ., Dept. of Foods & Nutrition, Corvallis. *Prof. & Head of Dept.* (5, 1940)

FINERTY, JOHN C., Ph.D. U. of Miami Sch. of Med., Dept. of Anatomy, Coral Gables, Fla. *Prof. and and Chmn.; Asst. Dean.* (1, 1947)

FINGL, EDWARD G., Ph.D. U. of Utah, Dept. of Pharmacol., Salt Lake City 1. *Asst. Prof.* (3, 1953)

FINK, KAY, Ph.D. VA Hosp., Long Beach, Calif. *Research Biochemist; Assoc. Clin. Prof. of Biophysics, UCLA.* (2, 1953)

FINK, MARY A., Ph.D. U. of Colorado Sch. of Med., Denver 22. *Asst. Prof. of Microbiol.* (6, 1955)

FINK, ROBERT M., Ph.D. U. of California Med. Ctr., Los Angeles 24. *Assoc. Prof., Physiol. Chem.* (2, 1953)

FINKEL, MIRIAM P., Ph.D. Argonne Natl. Lab., Lemont, Ill. *Assoc. Scientist.* (4, 1958)

FINLAND, MAXWELL, M.D. Boston City Hosp., Boston, Mass. *Assoc. Prof. of Med., Harvard Med. Scb.* (6, 1941)

FINNEGAN, J. K., Ph.D. Med. Coll. of Virginia, Richmond 19. *Assoc. Prof. of Pharmacol.* (3, 1947)

FIRMINGER, HARLAN I., M.D. U. of Maryland Sch. of Med., Baltimore 1. *Prof. of Patbology and Head of Dept.* (4, 1954)

FIROR, WARFIELD MONROE, M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. of Surgery, Johns Hopkins U.* (1, 1932)

FISCHEL, E. E., M.D., Sc.D.Med. Bronx Hosp., New York City 56. *Dir., Dept. Med.; Assoc. Clin. Prof. Med., Albert Einstein Coll. Med.* (6, 1948)

FISCHER, EDMOND H., Ph.D. U. of Washington Sch. of Med., Dept. of Biochem., Seattle 5. *Assoc. Prof.* (2, 1955)

FISCHER, ERNST, M.D. Med. Coll. of Virginia, Richmond. *Prof. of Physiol.* (1, 1936)

FISCHER, HERMANN O. L., Ph.D. U. of California, Dept. of Biochem., Berkeley 4. *Prof. Emer.* (2R, 1940)

FISCHER, MARIE A., Ph.D., M.D. South Side Hosp., South 20th and Jane Sts., Pittsburgh 3, Pa. (2, 1954)

FISCHER, MARTIN H., M.D. U. of Cincinnati, 107 Physics Bldg., Cincinnati 21, Ohio. *Prof. Emer. of Physiol.* (1, 1901; 2R, 1919)

FISHBERG, ELLA H., M.D. Beth Israel Hosp., Pearlman Pl., New York City 3. *Dir., Biochem. Lab.* (2, 1931)

FISHER, A. M., Ph.D. Connaught Labs., Toronto, Canada. *Asst. Dir.; Secy., Insulin Comm.; Assoc. Prof. Physiol. Hyg., U. Toronto.* (2, 1944)

FISHER, BERNARD, M.D. U. of Pittsburgh Med. Sch., Pittsburgh 13, Pa. *Assoc. Prof. of Surgery; Dir., Lab. of Exptl. Surgery.* (1, 1956)

FISHER, EDWIN R., M.D. VA Hosp., Pittsburgh 40, Pa. *Chief, Lab. Serv.* (4, 1954)

FISHER, HANS, Ph.D. New Jersey Agric. Expt. Sta., Rutgers U., New Brunswick, N. J. *Assoc. Prof. of Poultry Nutrition.* (5, 1958)

FISHER, HETTIE HUGHES, Ph.D. 2311 Jefferson Ave., Paducah, Ky. (2, 1946)

FISHER, KATHERINE H., Ph.D. Pennsylvania State U. Coll. of Home Econ., University Park. *Assoc. Prof. of Foods and Nutrition.* (5, 1958)

FISHER, KENNETH C., Ph.D. U. of Toronto, Dept. of Zool., Toronto, Canada. *Prof. and Dept. Chmn.* (1, 1940)

FISHMAN, ALFRED P., M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Med.; Dir., Cardioresp. Lab.* (1, 1952)

FISHMAN, WILLIAM H., Ph.D. Tufts U. Sch. of Med., Boston 11, Mass. *Research Prof., Biochem. and Nutrition.* (2, 1947)

FISKE, CYRUS H., M.D. 1155 Massachusetts Ave., Lexington 73, Mass. *Prof. Emer. of Biol. Chem., Harvard U.* (2R, 1914)

FITCH, FRANK W., M.D. U. of Chicago, Dept. of Pathol., Chicago 37, Ill. *PHS Post-doctoral Fellow.* (4, 1957)

FITZGERALD, MABEL P. 12 Crick Rd., Oxford, England. (1R, 1913)

FITZGERALD, PATRICK JAMES, M.D. State U. of New York Coll. of Med., Dept. of Pathol., Brooklyn 3. *Prof. and Chmn.* (4, 1956)

FITZHUGH, O. GARTH, Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D. C. *Chief, Toxicity Br.* (3, 1940)

FLAVIN, MARTIN, M.D., Ph.D. Natl. Heart Inst., Enzyme Sec., NIH, Bethesda 14, Md. (2, 1955)

FLEISCHMANN, WALTER, M.D., Ph.D. VA Ctr., Mountain Home, Tenn. *Chief, Lab. Serv.* (1, 1940)

FLEISHER, GERARD A., Ph.D. Mayo Clinic,

Rochester, Minn. *Assoc. Prof. of Biochem., Mayo Fndn., U. of Minnesota.* (2, 1957)

FLEISHER, MOYER S., M.D. Jewish Hosp., 216 S. Kingshighway Blvd., St. Louis 10, Mo. (4, 1924; 6, 1932)

FLEXNER, LOUIS B., M.D. U. of Pennsylvania Sch. of Med., Dept. of Anatomy, Philadelphia 4. *Prof. and Chmn. of Dept.* (1, 1933; 2, 1948)

FLICK, JOHN A., M.D. U. of Pennsylvania Grad. Sch. of Med., Philadelphia 4. *Assoc. Prof. and Chmn., Dept. of Microbiol.* (6, 1949)

FLOCK, EUNICE V., Ph.D. Mayo Clinic, Rochester, Minn. *Prof. of Biochem., Mayo Fndn., U. of Minnesota.* (2, 1940)

FLORMAN, ALFRED L., M.D. North Shore Hosp., Manhasset, N. Y. *Dir. of Pediatrics.* (6, 1942)

FLOYD, CLEAVELAND, M.D., Sc.D. 55 Appleton St., Cambridge, Mass. *Consultant, Boston Health Dept.* (6, 1916)

FLOYD, NORMAN F., B.S. Inst. for Cancer Research, Fox Chase, Philadelphia 11, Pa. *Research Assoc.* (2, 1957)

FLYNN, EDWIN H., Ph.D. Eli Lilly & Co., Indianapolis 6, Ind. *Research Chemist.* (2, 1958)

FLYNN, JOSEPH E., M.D. U. of Missouri Med. Sch., Dept. of Pathol., Columbia. *Prof. & Chmn. of Dept.* (4, 1953)

FOA, PIERO PIO, Ph.D., M.D. Chicago Med. Sch., 710 S. Wolcott St., Chicago, Ill. *Prof. of Physiol. and Pharmacol.* (1, 1944)

FODDEN, JOHN HENRY, M.D. Mt. Sinai Hosp., Div. of Labs., Milwaukee 3, Wis. *Assoc. Dir. of Labs.* (4, 1956)

FODOR, PAUL J., Ph.D. New York Med. Coll., Biochem. Dept., 5th Ave. at 105th St., New York City 29. *Asst. Prof.* (2, 1954)

FOGG, LLOYD C., Ph.D. Pondville Hosp., Walpole, Mass. *Instr. in Pathol., Harvard Med. Sch.* (4, 1957)

FOLCH, JORDI, M.D. McLean Hosp., Waverley, Mass. *Dir. of Sci. Research; Prof. of Neurochem., Harvard Med. Sch.* (2, 1941)

FOLDES, FRANCIS F., M.D. Mercy Hosp., Pittsburgh 19, Pa. *Dir. of Anesthesiol.* (3, 1952)

FOLEY, GEORGE E., Sc.D. Children's Cancer Research Fndn., Boston 15, Mass. *Chief, Labs. of Microbiol.* (4, 1957)

FOLK, G. EDGAR, JR., Ph.D. State U. of Iowa, Dept. of Physiol., Iowa City. (1, 1953)

FOLK, J. E., Ph.D. Natl. Inst. of Dental Research, NIH, Bethesda 14, Md. *Research Assoc., Amer. Dental Assn.; Biochemist.* (2, 1958)

FOLKERS, KARL, Ph.D. Merck Sharp & Dohme Research Labs., Rahway, N. J. *Exec. Dir., Fundamental Research.* (2, 1947; 5, 1954)

FOLLENSBY, EDNA M., Ph.D. 80 E. Concord St., Boston, Mass. *Research Assoc., Boston U. Sch. of Med.* (6, 1933)

FOLLIS, RICHARD H., JR., M.D. Armed Forces Inst. of Pathol., Walter Reed Army Med. Ctr., Washington 25, D.C. (4, 1942)

FOLTZ, ELWOOD L., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof. in Med.; Assoc. in Clin. Pharmacol.* (1, 1952; 3, 1955)

FONG, JACOB, Ph.D. 2640 Derby St., Berkeley 5, Calif. *Assoc. Prof. of Bacteriol., U. of California.* (6, 1951)

FONTAINE, THOMAS D., Ph.D. Natl. Science Fndn., Washington 25, D.C. *Program Dir. for Fellowships.* (2, 1946)

FOOT, NATHAN CHANDLER, M.D. Cornell U. Med. Coll., New York City 21. *Consultant.* (4R, 1924)

FORAKER, ALVAN GLENN, M.D. Baptist Mem. Hosp., Jacksonville, Fla. *Pathologist; Physician.* (4, 1954)

FORBES, ALEXANDER, M.D. Harvard U. Biol. Labs., Cambridge, Mass. *Prof. Emer. of Physiol.* (1, 1910)

FORBES, ERNEST B., Ph.D. 500 W. Fairmount Ave., State College, Pa. *Prof. Emer. Animal Nutrition, Pa. State U.* (1R, 1917; 5R, 1933)

FORBES, HENRY S., M.D. 71 Forest St., Milton 86, Mass. (1R, 1931)

FORBES, JOHN C., Ph.D. Med. Coll. of Virginia, Richmond. *Research Prof. of Biochem.* (2, 1937)

FORBES, RICHARD M., Ph.D. U. of Illinois, Div. of Animal Nutrition, Urbana. *Prof.* (5, 1953)

FORBES, WILLIAM H., Ph.D., M.D. Harvard Sch. of Public Health, Boston 15, Mass. (1, 1943)

FORMAL, SAMUEL B., Ph.D. Walter Reed Army Inst. of Research, Washington 12, D.C. *Chief, Dept. of Applied Immunol.* (6, 1957)

FORNEY, ROBERT B., Ph.D. Indiana U. Sch. of Med., Indianapolis 7. *Assoc. Prof. of Toxicol.* (3, 1950)

FORSCHER, BERNARD K., Ph.D. U. of Kansas City Sch. of Dent., Kansas City, Mo. *Assoc. Prof. & Chmn. of Biochem.* (4, 1958)

FORSTER, FRANCIS M., M.D. Georgetown U. Sch. of Med., Dept. of Neurol., Washington 7, D.C. *Prof. and Dir. of Dept.* (1, 1948)

FORSTER, ROBERT ELDER, II, M.D. 721 Millbrook Lane, Haverford, Pa. *Prof. of Physiol., U. of Pa. Grad. Sch. of Med.* (1, 1951)

FORSTER, ROY P., Ph.D. Dartmouth Coll., Dept. of Zool., Hanover, N. H. *Prof.* (1, 1950)

FORTIER, CLAUDE, M.D., Ph.D. Baylor U. Coll. of Med., Houston, Tex. *Assoc. Prof. Physiol.; Dir., Blue Bird Neurol. Res. Labs.* (1, 1957)

FOSDICK, LEONARD S., Ph.D. 311 E. Chicago Ave., Chicago, Ill. *Prof. of Chem., Northwestern U.* (2, 1944)

FOSTER, G. L., Ph.D. R. 1, Raymond, N. H. *Emer. Prof. of Biochem., Columbia U., Coll. of P. & S.* (2R, 1923)

FOSTER, HARRY E., M.D. c/o American Express, 11 Rue Scribe, Paris 9^{e}, France. (6R, 1913)

FOSTER, JACKSON W., Ph.D. U. of Texas, Dept. of Bacteriol., Austin 12. *Prof.* (2, 1946)

FOSTER, JOSEPH F., Ph.D. Purdue U., Dept. of Chem., Lafayette, Ind. *Prof.* (2, 1952)

FOSTER, ROBERT H. K., Ph.D., M.D. 301 E. Jefferson St., Franklin, Ind. *Practicing Physician.* (3, 1944)

FOSTER, WILLIAM C., M.S. Misericordia Hosp., Philadelphia 43, Pa. *Dir., Chem. Lab.; Consulting Physiologist.* (1, 1952)

FOTHERGILL, LEROY D., M.D. Fort Detrick, Frederick, Md. (6, 1952)

FOULKS, JAMES G., Ph.D., M.D. U. of British Columbia, Dept. of Pharmacol., Vancouver, Canada. *Prof. and Head of Dept.* (1, 1957; 3, 1956)

FOWLER, WARD S., M.D. Mayo Fndn., Dept. of Physiol., Rochester, Minn. *Prof.* (1, 1950)

FOX, CHARLES L., JR., M.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City 29. *Assoc. Prof., Surg. & Biochem.* (1, 1951; 6, 1951)

FOX, JACK J., Ph.D. Sloan-Kettering Inst. for Cancer Research, New York City 21. *Assoc.* (2, 1954)

FOX, JOHN P., M.D., Ph.D. Tulane U. Sch. of Med., 1430 Tulane Ave., New Orleans, La. *Prof. of Epidemiol.* (6, 1953)

FOX, M. R. SPIVEY, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, Lab. of Nutr. & Endocrinol., NIH, Bethesda 14, Md. *Biochemist.* (5, 1958)

FOX, SIDNEY W., Ph.D. Florida State U. Oceanographic Inst., Tallahassee. *Dir. and Prof.; Prof., Chem. Dept.* (2, 1946)

FRAENKEL, GOTTFRIED S., Ph.D. U. of Illinois, Urbana. *Prof. of Entomol.* (5, 1949)

FRAENKEL-CONRAT, HEINZ, M.D., Ph.D. U. of California, Biochem. & Virus Lab., Berkeley. *Prof. of Virol.* (2, 1942)

FRANCIS, THOMAS, JR., M.D. U. of Michigan Sch. of Public Health, Ann Arbor. *Chmn. & Henry Sewall U. Prof. Epidemiol.* (4, 1940; 6, 1930)

FRANK, KARL, Ph.D. Natl. Inst. of Neurological Diseases & Blindness, NIH, Bethesda 14, Md. *Neurophysiologist, Spinal Cord Sec.* (1, 1953)

FRANKE, FLORENT E., M.D. St. Louis U. Sch. of Med., St. Louis 10, Mo. *Assoc. Prof. of Physiol.* (1, 1934)

FRANTZ, IVAN D., JR., M.D. U. of Minnesota, Dept. of Med., Minneapolis 14. *Research Prof.* (2, 1953)

FRAPS, R. M., Ph.D. USDA, Agric. Research Serv., Beltsville, Md. *Physiologist.* (1, 1947)

FRASER, HAVELOCK F., M.D. Public Health Serv. Hosp., Lexington, Ky. *Asst. Dir., NIMH Addiction Res. Ctr.* (3, 1952)

FRAWLEY, JOHN P., Ph.D. Hercules Powder Co., Med. Dept., Wilmington 99, Del. *Toxicologist.* (3, 1953)

FREDETTE, VICTORIEN. U. of Montreal, Montreal, Canada. *Assoc. Prof. of Bacteriol.; Assoc. Dir., Inst. of Microbiol.* (6, 1948)

FREE, ALFRED H., Ph.D. Miles-Ames Research Lab., Elkhart, Ind. *Head of Biochem. Sec.* (2, 1946; 5, 1944)

FREEDBERG, A. STONE, M.D. Beth Israel Hosp., Boston 15, Mass. *Assoc. Dir., Med. Res. Dept.; Assoc. Prof. Med., Harvard.* (1, 1952)

FREEMAN, LESLIE WILLARD, Ph.D., M.D. Indiana U., Dept. of Surgery, Indianapolis. *Prof.; Dir., Surgical Exptl. Labs.* (1, 1944)

FREEMAN, MARIAN VIRGINIA, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chemist.* (3, 1958)

FREEMAN, NORMAN E., M.D. Stanford U. Med. Sch., San Francisco, Calif. (1, 1936)

FREEMAN, SMITH, M.D., Ph.D. Northwestern U. Sch. of Med., Chicago 11, Ill. *Prof. and Chmn., Dept. of Biochem.* (1, 1937)

FREGLY, MELVIN J., Ph.D. U. of Florida Coll. of Med., Gainesville. *Asst. Prof. of Physiol.* (1, 1955)

FREIMAN, DAVID G., M.D. Beth Israel Hosp., Dept. of Pathol., Boston 15, Mass. *Clin. Prof. of Pathol., Harvard Med. Sch.* (4, 1954)

FREIS, EDWARD D., M.D. Georgetown U. Hosp., Cardiovasc. Res. Lab., Washington, D. C. *Chief; Chief, Cardiovasc. Res. Lab. VA Hosp.* (3, 1955)

FRENCH, C. STACY, Ph.D. Carnegie Inst. of Washington, Stanford, Calif. *Dir., Dept. of Plant Biol.* (2, 1946)

FRENCH, CYRUS E., Ph.D. UNICEF, United Nations, New York City. *Chief, Nutrition.* (5, 1948)

FRENCH, DEXTER, Ph.D. Iowa State Coll., Dept. of Chem., Ames. *Prof.* (2, 1953)

FRENKEL, ALBERT W., Ph.D. U. of Minnesota, Dept. of Botany, Minneapolis 14. *Prof.* (2, 1957)

FRENKEL, JACOB KARL, M.D., Ph.D. U. of Kansas Sch. of Med., Kansas City 12. *Assoc. Prof. of Pathol.* (4, 1953)

FRETER, ROLF, Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Asst. Prof. of Microbiol.* (6, 1958)

FREUND, JULES, M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Chief, Lab. of Immunol.* (6, 1924)

FREY, CHARLES N., Ph.D., D.Sc. 45 Cambridge Rd., Scarsdale, N. Y. *Lect., M.I.T.; Consultant.* (5, 1948)

FREYBURGER, WALTER A., JR., Ph.D. The Upjohn Co., Kalamazoo, Mich. *Sec. Head, Pharmacol.* (3, 1951)

FREYGANG, WALTER H., JR., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Surgeon, PHS.* (1, 1955)

FRIED, GEORGE H., Ph.D. Beth Israel Hosp., New York City 3. *Research Assoc., Levy Labs.* (4, 1957)

FRIED, JOSEF, Ph.D. Squibb Inst. for Med. Research, New Brunswick, N. J. *Dept. Head, Div. of Organic Chem.* (2, 1948)

FRIEDBERG, FELIX, Ph.D. Howard U. Med. Sch., Dept. of Biochem., Washington 1, D. C. *Asst. Prof.* (2, 1951)

FRIEDEMANN, THEODORE E., Ph.D. USA Med. Nutrition Lab., Fitzsimons Army Hosp., Denver 8, Colo. *Sci. Dir.* (2, 1925; 5, 1952)

FRIEDEN, EARL, Ph.D. Florida State U., Dept. of Chem., Tallahassee. *Assoc. Prof.* (2, 1956)

FRIEDEN, EDWARD H., Ph.D. Boston Dispensary, 25 Bennet St., Boston 11, Mass. *Res. Coordinator; Asst. Prof. of Biochem., Tufts U.* (2, 1950)

FRIEDEWALD, WILLIAM F., M.D. Emory U. Sch. of Med., Atlanta 22, Ga. *Assoc. Prof. of Med.* (4, 1941)

FRIEDGOOD, HARRY B., M.D. U. of California Med. Ctr., Dept. of Med., San Francisco 22. *Lect.* (1, 1936)

FRIEDKIN, MORRIS ENTON, Ph.D. Tufts U. Sch. of Med., Dept. of Pharmacol., Boston 11, Mass. *Prof. & Head of Dept.* (2, 1951)

FRIEDMAN, LEO, Ph.D. Food & Drug Admin., Bur. Biol. & Physical Sci., Dept. of HEW, Washington 25, D. C. *Chief, Biol. Br.* (5, 1958)

FRIEDMAN, M. H. F., Ph.D. Jefferson Med. Coll., Dept. of Physiol., Philadelphia 7, Pa. *Prof. and Head of Dept.* (1, 1941)

FRIEDMAN, MAURICE H., Ph.D., M.D. 6 Pinehurst Circle, Washington 15, D. C. (1, 1929)

FRIEDMAN, MEYER, M.D. Harold Brunn Inst. for Cardiovasc. Research, Mt. Zion Hosp., San Francisco, Calif. *Dir.* (1, 1947)

FRIEDMAN, NATHAN B., M.D. Cedars of Lebanon

Hosp., Div. of Labs., 4833 Fountain Ave., Los Angeles, Calif. *Dir.* (4, 1942)

FRIEDMAN, SYDNEY M., M.D., Ph.D. U. of British Columbia, Dept. of Anatomy, Vancouver, Canada. *Prof. and Head of Dept.* (1, 1947)

FRIEND, CHARLOTTE, Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc.; Asst. Prof., Sloan-Kettering Div., Cornell U.* (6, 1957)

FRIEND, DALE G., M.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. in Med.; Sr. Assoc. in Med., Peter Bent Brigham Hosp.* (3, 1955)

FRISELL, WILHELM R., Ph.D. U. of Colorado Sch. of Med., Denver 20. *Assoc. Prof. of Biochem.* (2, 1956)

FRITTS, HARRY W., JR., M.D. Bellevue Hosp., Cardio-Pulmonary Lab., New York City 16. *Asst. Prof. of Med., Columbia U.* (1, 1958)

FRITZ, IRVING B., D.D.S., Ph.D. U. of Michigan, Dept. of Physiol., Ann Arbor. *Asst. Prof.* (1, 1956)

FRITZ, JAMES C., B.S. Dawe's Labs., Inc., 4800 S. Richmond St., Chicago 32, Ill. *Dir. of Nutrition Research.* (5, 1951)

FROHMAN, CHARLES E., Ph.D. Lafayette Clinic, Detroit 7, Mich. *Biochemist.* (2, 1956)

FROST, DOUGLAS VAN ANDEN, Ph.D. Abbott Labs., North Chicago, Ill. *Head of Nutritional Research.* (2, 1946; 5, 1947)

FRUMIN, M. JACK, M.D. Columbia U., Coll. of P. & S., New York City 32. *Asst. Prof. of Anesthesiol.* (3, 1957)

FRUTON, J. S., Ph.D. Yale U. Sch. of Med., Dept. of Biochem., New Haven 11, Conn. *Chmn.; Eugene Higgins Prof. Biochem.* (2, 1938)

FRY, WILLIAM J., M.S. U. of Illinois, Biophysical Research Lab., Urbana. *Head of Lab.; Research Prof.* (1, 1954)

FU, SHOU-CHENG JOSEPH, Ph.D. Children's Med. Ctr., Boston 15, Mass. *Research Assoc.* (2, 1957)

FUGO, NICHOLAS W., Ph.D., M.D. U. of Chicago, Dept. of Obstetrics and Gynecol., Chicago 37, Ill. *Asst. Prof.* (3, 1944)

FUHRMAN, FREDERICK A., Ph.D. Stanford U., Stanford, Calif. *Prof. of Physiol.* (1, 1946)

FUKUSHIMA, DAVID K., Ph.D. Sloan-Kettering Inst. for Cancer Research, New York City 21. *Assoc., Steroid Biochem.* (2, 1952)

FULD-CONDERMAN, MARIA, Ph.D. 4916 Bayard St. Pittsburgh 13, Pa. (2, 1953)

FULLER, HENRY SHEPARD, M.D. Walter Reed Army Inst. of Research, Dept. of Rickettsial Diseases, Washington 12, D.C. *Chief.* (6, 1956)

FULLER, JOHN L., Ph.D. Box 847, Bar Harbor, Maine. *Staff Scientist, Jackson Mem. Lab.* (1, 1954)

FULTON, GEORGE P., Ph.D. Boston U., Dept. of Biol., 675 Commonwealth Ave., Boston, Mass. *Prof. and Chmn. of Dept.* (1, 1950)

FULTON, JOHN D., Ph.D. USAF Sch. of Aviation Med., Dept. of Microbiol., Randolph AFB, Tex. *Acting Chief of Dept.; Lt. Col., USAF.* (6, 1958)

FULTON, JOHN FARQUHAR, Ph.D., M.D. Yale U. Sch. of Med., New Haven, Conn. *Sterling Prof. of History of Med.* (1, 1925)

FUNDERBURK, WILLIAM H., Ph.D. Miles-Ames Research Labs., Elkhart, Ind. *Pharmacologist; Lect. U. of Illinois Med. Sch.* (3, 1953)

FUNK, CASIMIR, D.Sc., Ph.D. 186 Riverside Drive, New York City 24. *Pres., Funk Fndn. for Med. Research.* (2R, 1921; 5, 1958)

FUORTES, M. G. F., M.D. Natl. Inst. of Neurological Diseases & Blindness, Ophthalmology Br., NIH, Bethesda 14, Md. (1, 1952)

FURCHGOTT, ROBERT F., Ph.D. State U. of New York Coll. of Med., Dept. of Pharmacol., Brooklyn 3. *Prof. and Chmn. of Dept.* (2, 1948; 3, 1952)

FURMAN, R. H., M.D. Oklahoma Med. Res. Fndn., Oklahoma City 4. *Assoc. Dir. Res.; Assoc. Prof. Res. Med., U. Okla.* (1, 1958)

FURST, ARTHUR, Ph.D. Stanford U. Sch. of Med., Stanford, Calif. *Assoc. Prof. of Pharmacol.* (3, 1957)

FURTH, JACOB, M.D. Children's Cancer Research Fndn., 35 Binney St., Boston 15, Mass. *Assoc. Dir. of Research.* (4, 1932)

GABRIO, BEVERLY W., Ph.D. King County Central Blood Bank, Seattle, Wash. *Dir. Biochem. Res.; Lect. Med., U. Washington.* (2, 1954)

GABY, WILLIAM L., Ph.D. Hahnemann Med. Coll., Div. of Microbiol., Philadelphia 2, Pa. *Assoc. Prof.* (6, 1957)

GADDUM, JOHN H., Sc.D. Inst. of Animal Physiol., Babraham, Nr. Cambridge, England. *Dir.* (3, 1950)

GAEBLER, OLIVER H., Ph.D., M.D. Henry Ford Hosp., Detroit 2, Mich. *Head, Dept. Biochem., Edsel B. Ford Inst. for Med. Res.* (2, 1927)

GAFFRON, HANS, Ph.D. U. of Chicago, Research Insts., Chicago 37, Ill. *Prof. of Biochem., Dept. of Biochem. and Research Insts.* (2, 1941)

GAGGE, ADOLF P., Ph.D. USAF, Office of Sci. Research, Washington 25, D.C. *Col., USAF.* (1, 1939)

GAINES, SIDNEY, Ph.D. Walter Reed Army Inst. of Research, Washington 12, D.C. *Chief, Dept. Microbiol., Spec. Operations Div.* (6, 1957)

GAL, EMERY M., Ph.D. U. of California Med. Ctr., Dept. of Pharmacol., Los Angeles 24. *Assoc. Prof.* (2, 1955)

GALAMBOS, ROBERT, Ph.D., M.D. Walter Reed Army Inst. of Research, Dept. of Neurophysiol., Washington 12, D.C. *Chief.* (1, 1942)

GALDSTON, MORTON, M.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (1, 1950)

GALL, EDWARD A., M.D. Cincinnati Gen. Hosp., Cincinnati, Ohio. *Prof. of Patbol., U. of Cincinnati Coll. of Med.* (4, 1941)

GALLAGHER, THOMAS F., Ph.D. Sloan-Kettering Inst., New York City 21. *Chief, Divs. Steroid Metab. and Biochem.* (2, 1932)

GALLUP, WILLIS D., Ph.D. Oklahoma State U., Stillwater. *Chemist and Prof. of Agric. Chem.* (2, 1932)

GALSTON, ARTHUR W., Ph.D. Yale U., Josiah Willard Gibbs Res. Lab., Dept. of Botany, New Haven, Conn. *Prof. of Plant Physiol.* (2, 1958)

GAMBLE, JAMES L., M.D., Sc.D. 33 Edgehill Rd., Brookline, Mass. *Prof. Emer. of Pediatrics, Harvard Med. Sch.* (2R, 1922; 5R, 1933)

GANONG, WILLIAM FRANCIS, M.D. U. of California Sch. of Med., Dept. of Physiol., San Francisco 22. *Asst. Prof.* (1, 1957)

GANTT, W. HORSLEY, M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. Psychiatry; Dir., Pavlovian Lab.* (1, 1935; 3, 1954)

GANZ, AARON, Ph.D. U. of Tennessee Med. Units, Memphis 3. *Assoc. Prof. of Pharmacol.* (3, 1957)

GARB, SOLOMON, M.D. Albany Med. Coll., Albany, N.Y. *Assoc. Prof. of Pharmacol.* (3, 1951)

GARBAT, ABRAHAM L., M.D. 103 E. 78th St., New York City. *Consultant Physician, Lenox Hill Hosp.* (6, 1913)

GARDNER, ERNEST, M.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Prof. of Anatomy.* (1, 1949)

GARDOCKI, JOSEPH F., Ph.D. Squibb Inst. for Med. Research, New Brunswick, N.J. *Research Pharmacologist.* (3, 1957)

GARNER, RAYMOND L., Ph.D. Seton Hall Coll. of Med. and Dent., Dept. of Biochem., Jersey City 4, N.J. *Prof. and Chmn.* (2, 1947)

GARREN, HENRY W., Ph.D. North Carolina State Coll., Raleigh. *Assoc. Prof. of Poultry Endocrinol.* (1, 1957)

GARST, JOSEPHINE B., Ph.D. VA Hosp., 7300 York Rd., Cleveland 30, Ohio. *Research Biochemist.* (2, 1955)

GARVEY, JUSTINE S., Ph.D. California Inst. of Tech., Div. of Chem & Chem. Engin., Pasadena 4. *Sr. Research Fellow.* (6, 1956)

GASSER, HERBERT S., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Dir., Emer.* (1, 1915; 3, 1924)

GASSNER, FRANK X., D.V.M. Colorado State U., Dept. of Chem., Fort Collins. *Prof. of Endocrine Research.* (1, 1947)

GASTEIGER, EDGAR L., JR., Ph.D. U. of Rochester Sch. of Med. and Dent., Dept. of Physiol., Rochester, N.Y. *Asst. Prof.* (1, 1954)

GATES, OLIVE, M.D. Cancer Research Inst., New England Deaconess Hosp., 194 Pilgrim Rd., Boston, Mass. *Assoc. Pathologist.* (4, 1940)

GAUDINO, MARIO, M.D., Ph.D. Abbott Labs. Internatl. and Universal, Merchandise Mart Plaza, Chicago 54, Ill. *Med. Dir.* (1, 1951)

GAUER, OTTO H., M.D. W.G. Kerckhoff Inst. d. Max-Planck-Gesellschaft, Bad Nauheim, West Germany. *Prof.* (1, 1954)

GAUNT, ROBERT, Ph.D. Ciba Pharmaceutical Products, Inc., Summit, N.J. *Dir., Biologic Research.* (1, 1939)

GAWRON, OSCAR, Ph.D. Duquesne U., Dept. of Chem., Pittsburgh 19, Pa. *Prof.* (2, 1956)

GAY, LESLIE N., M.D., D.Sc. 1114 St. Paul St., Baltimore, Md. *Assoc. Prof. Med., Johns Hopkins U.; Dir., Allergy Clinic, Johns Hopkins Hosp.* (6, 1927)

GEARY, JOHN R., JR., M.D., Ph.D. 405 Spruce St., Mill Valley, Calif. (4, 1954)

GEBHARDT, LOUIS P., M.D. U. of Utah Coll. of Med., Dept. of Bacteriol., Salt Lake City 12. *Prof. and Head of Dept.* (6, 1954)

GEDDES, WILLIAM F., Ph.D. U. of Minnesota, Dept. of Agric. Biochem., St. Paul 1. *Prof. and Head of Dept.* (2, 1953)

GEIGER, ERNEST, M.D., Ph.D. 4233 Don Ortega Pl., Los Angeles 8, Calif. *Prof. of Biochem. & Nutr., U. Southern Calif. Med. Sch.* (5, 1950)

GEILING, E. M. K., M.D., Ph.D. U. of Chicago, Dept. of Pharmacol., Chicago 37, Ill. *Prof. Emer.* (1R, 1933; 2R, 1927; 3, 1925)

GEIMAN, QUENTIN M., Ph.D. Stanford U. Sch. of Med., San Francisco, Calif. *Prof. of Prev. Med. (Tropical Public Health).* (4, 1955)

GELFAN, SAMUEL, Ph.D. 1249 Fifth Ave., New York City 29. *Research Assoc. in Neurol. and Neurosurgery, New York Med. Coll.* (1, 1930)

GELL, C. F., M.D., D.Sc.(Med.) Dept. of the Navy, Office of Naval Research, Washington 25, D.C. *Spec. Asst. for Med. & Allied Sci.* (1, 1955)

GELLHORN, ALFRED, M.D. Columbia U. Coll. of P. & S., Dept. of Med., New York City 32. *Prof.* (3, 1946)

GELLHORN, ERNST, M.D., Ph.D. 2 Fellowship Circle, Santa Barbara, Calif. (On leave from U. of Minnesota, *Prof. of Neurophysiol.*) (1, 1930)

GEMMILL, CHALMERS L., M.D. U. of Virginia Med. Sch., Charlottesville. *Prof. of Pharmacol.* (1, 1928; 2, 1935; 3, 1946)

GENGOZIAN, NAZARETH, Ph.D. Oak Ridge Natl. Lab., Biology Div., P.O. Box Y, Oak Ridge, Tenn. (6, 1958)

GEORGE, PHILIP, Ph.D. U. of Pennsylvania, John Harrison Lab. of Chem., Philadelphia 4. *Research Prof. of Biophysical Chem.* (2, 1958)

GEORGE, ROBERT, Ph.D. U. of California Med. Ctr., Dept. of Pharmacol., Los Angeles 24. *Asst. Prof.* (3, 1957)

GERARD, R. W., Ph.D., M.D. U. of Michigan, Mental Health Research Inst., Ann Arbor. *Prof. of Neurophysiol.* (1, 1927)

GERGELY, JOHN, M.D., Ph.D. Mass. Gen. Hosp., Cardiac Biochem. Res. Lab., Boston. *Assoc. Biochemist; Res. Assoc. in Med., Harvard.* (2, 1952)

GERHEIM, EARL B., Ph.D. Sherman Labs., 5031 Grandy, Detroit 11, Mich. *Dir., Bio-Sci. Res.* (1, 1950; 6, 1950)

GERMUTH, FREDERICK G., JR., M.D. Charlotte Mem. Hosp., 1400 Scott Ave., Charlotte, N.C. (4, 1956; 6, 1954)

GERSCHMAN, REBECA, Ph.D. U. of Rochester Sch. of Med. and Dent., Dept. of Physiol., Rochester 20, N.Y. *Asst. Prof.* (1, 1955)

GERSH, ISIDORE, Ph.D. U. of Chicago, Chicago 37, Ill. *Prof. of Anatomy.* (4, 1947)

GERSHBERG, HERBERT, M.D. New York U. Coll. of Med., New York City. *Asst. Prof. of Med. Physiol.* (1, 1954)

GERSHOFF, STANLEY N., Ph.D. Harvard Sch. of Public Health, Dept. of Nutrition, Boston 15, Mass. *Asst. Prof.* (5, 1958)

GERSTEN, JEROME W., M.D. U. of Colorado Med. Ctr., Denver 20. *Prof. and Head, Physical Med. and Rehabilitation.* (1, 1951)

GERSTNER, HERBERT B., M.D. USAF Sch. of Aviation Med., Randolph AFB, Tex. *Assoc. Prof. of Radiobiol.* (1, 1956)

GERTNER, SHELDON B., Ph.D. Seton Hall Coll. of Med. and Dent., Dept. of Pharmacol., Jersey City 4, N.J. *Asst. Prof.* (3, 1958)

GESLER, ROBERT M., Ph.D. Baxter Labs., Inc., Morton Grove, Ill. *Chief, Pharmacol. Sec.* (3, 1957)

GEST, HOWARD, Ph.D. Western Reserve U. Sch. of Med., Dept. of Microbiol., Cleveland 6, Ohio. *Assoc. Prof.* (2, 1952)

GETTLER, ALEXANDER O., Ph.D. 400 E. 29th St., New York City 16. *Prof. Emer. N.Y.U.; Toxicologist, Chief Med. Examiner's Office.* (2R, 1916)

GEY, GEORGE OTTO, M.D. Johns Hopkins U., Baltimore 5, Md. *Dir., Finney-Howell Cancer Res. Lab.; Asst. Prof. of Surgery.* (1, 1940)

GEYER, ROBERT P., Ph.D. Harvard Sch. of Public Health, Dept. of Nutrition, Boston, Mass. *Assoc. Prof.* (5, 1950)

GEZON, HORACE MARTIN, M.D. U. of Pittsburgh Grad. Sch. of Public Health, Pittsburgh 13, Pa. *Prof. of Epidemiol. & Microbiol.* (6, 1956)

GIARMAN, NICHOLAS J., Ph.D. Yale U. Med. Sch., Dept. of Pharmacol., New Haven, Conn. *Assoc. Prof.* (3, 1953)

GIBBARD, J., S.M. Dept. of Natl. Health and Welfare, Ottawa, Canada. *Dir., Lab. of Hygiene.* (6, 1946)

GIBBS, FREDERIC ANDREWS, M.D. 720 N. Michigan Ave., Suite 610, Chicago, Ill. (1, 1935)

GIBBS, MARTIN, Ph.D. Cornell U., Dept. of Biochem., Ithaca, N. Y. (2, 1953)

GIBBS, OWEN STANLEY, M.D. P. O. Box 5387, Whitehaven, Memphis 16, Tenn. (3, 1930)

GIBSON, DAVID M., M.D. Indiana U. Sch. of Med., Dept. of Biochem., Indianapolis 7. *Assoc. Prof.* (2, 1956)

GIBSON, ROBERT BANKS, Ph.D. University Hosp., Iowa City, Iowa. *Emer. Prof. of Biochem., State U. of Iowa.* (2R, 1906)

GILBERT, JAMES B., M.D. U. of California, Dept. of Biochem., Berkeley 4. *Estab. Invest., Amer. Heart Assn.* (2, 1955)

GILLIS, MARVIN B., Ph.D. Internatl. Minerals and Chemical Corp., Skokie, Ill. *Dir. of Research.* (5, 1952)

GILMAN, ALFRED, Ph.D. Albert Einstein Coll. of Med., Dept. of Pharmacol., New York City 61. *Prof. and Chmn. of Dept.* (1, 1935; 3, 1934)

GILSON, ARTHUR S., JR., Ph.D. Washington U. Med. Sch., St. Louis 10, Mo. *Assoc. Prof. of Physiol.* (1, 1927)

GILVARG, CHARLES, Ph.D. New York U.-Bellevue Med. Ctr., Dept. of Biochem., New York City 16. *Asst. Prof.* (2, 1955)

GINSBERG, HAROLD S., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof. of Prev. Med.; Asst. Prof. of Med.* (6, 1951)

GITHENS, THOMAS S., M.D. The Cambridge, Philadelphia 44, Pa. (1R, 1915)

GITLIN, DAVID, M.D. Children's Med. Ctr., Boston 15, Mass. *Assoc. Physician; Asst. Prof. of Pediatrics, Harvard Med. Sch.* (6, 1958)

GIVENS, MAURICE H., Ph.D. Box 5116, Biltmore, N. C. (1R, 1917; 2R, 1915)

GJESSING, ERLAND CHENEY, Ph.D. U. of Vermont Coll. of Med., Dept. of Biochem., Burlington. *Assoc. Prof.* (2, 1948)

GLASER, ROBERT JOY, M.D. U. of Colorado Sch. of Med., Denver 20. *Dean and Prof. of Med.* (4, 1954)

GLASS, GEORGE B. JERZY, M.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City. *Assoc. Prof. of Med.* (1, 1953)

GLASS, HOWARD G., M.D., Ph.D. Miles-Ames Research Lab., Elkhart, Ind. *Pharmacologist.* (3, 1947)

GLASSMAN, JEROME M., Ph.D. Wyeth Inst. for Med. Res., Dept. Pharmacol., Box 8299, Philadelphia 1, Pa. *Sr. Res. Pharmacologist.* (3, 1956)

GLAZKO, ANTHONY J., Ph.D. Parke, Davis & Co., Research Labs., Detroit 32, Mich. *Research Biochemist.* (1, 1942)

GLEISER, CHESTER A., V.M.D., M.P.H. Walter Reed Army Med. Ctr., Fort Detrick, Md. *Veterinary Pathologist.* (4, 1958)

GLICK, DAVID, Ph.D. U. of Minnesota, Dept. of Physiol. Chem., 180 Lyon Labs., Minneapolis 14. *Prof.* (2, 1936)

GLICKMAN, NATHANIEL, M.S. 970 So. Shore Dr., Miami Beach, Fla. (1, 1947)

GLINOS, ANDRE D., M.D. Walter Reed Army Inst. of Research, Washington 12, D. C. *Chief, Growth Physiol. Lab.* (1, 1957)

GLUCKMAN, MELVYN I., Ph.D. VA Hosp., Pittsburgh 6, Pa. (3, 1954)

GOCHENOUR, WILLIAM S., JR., V.M.D. Walter Reed Army Med. Ctr., Med. Unit, Fort Detrick, Md. (6, 1950)

GODDARD, DAVID R., Ph.D. U. of Pennsylvania, Botanical Labs., Philadelphia 4. *Prof. of Botany.* (2, 1953)

GODDARD, JAMES W., M.D. Jefferson Med. Coll., Dept. of Exptl. Med., Philadelphia 7, Pa. *Research Pathologist.* (4, 1956)

GODWIN, JOHN T., M.D. St. Joseph's Infirmary, 272 Courtland St., N. E., Atlanta, Ga. *Dir. of Labs.* (4, 1954)

GOEBEL, WALTHER F., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1929; 6, 1937)

GOETTSCH, MARIANNE, Ph.D. U. of Puerto Rico Sch. of Med., San Juan 22. *Assoc. Prof. of Biochem. and Nutrition.* (2, 1933; 5, 1941)

GOETZL, FRANZ R., Ph.D., M.D. 2622 Piedmont Ave., Berkeley 4, Calif. (1, 1947)

GOLD, HARRY, M.D. Cornell U. Med. Coll., New York City 21. *Prof. of Clin. Pharmacol.* (3, 1927)

GOLDBAUM, LEO R., Ph.D. Tacoma Gen. Hosp., Tacoma 5, Wash. (3, 1952)

GOLDBERG, HARRY, M.D. Hahnemann Med. Coll. and Hosp., Philadelphia 2, Pa. *Asst. Prof. of Med.; Dir., Cardiopulmonary Lab.* (1, 1955)

GOLDBERG, LEON I., Ph.D., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Clin. Assoc.* (3, 1953)

GOLDBLATT, HARRY, M.D. Mt. Sinai Hosp., Cleveland 6, Ohio. *Dir. of Labs.; Prof. Exptl. Pathol., Western Reserve U.* (1, 1945; 4, 1927)

GOLDEN, ALFRED, M.D. 26764 York Rd., Huntington Woods, Mich. (4, 1947)

GOLDFARB, A. R., Ph.D. Chicago Med. Sch., Chicago 12, Ill. *Asst. Prof.* (2, 1955)

GOLDFEDER, ANNA, D.Sc. 920 Riverside Dr., New York City 32. *Sr. Biologist, Dept. of Hosps., City of N. Y.; Res. Assoc., N. Y. U.* (1, 1950)

GOLDFORB, A. J., Ph.D. City Coll. of New York, New York City. *Prof. Emer. of Biol.* (1, 1930)

GOLDIE, HORACE, M.D., D.T.M. Meharry Med. Coll., Nashville 8, Tenn. *Prof. of Surgery (Exptl. Oncol.) and Dir.* (6, 1943)

GOLDIN, ABRAHAM, Ph.D. Natl. Cancer Inst., Lab. of Chem. Pharmacol., NIH, Bethesda 14,

Md. *Pharmacologist.* (1, 1951; 3, 1956)

GOLDMAN, DAVID E., Ph.D. Naval Med. Research Inst., Bethesda 14, Md. (1, 1951)

GOLDMAN, DEXTER S., Ph.D. VA Hosp., Madison 5, Wis. *Chief Biochemist; Asst. Prof., Inst. for Enzyme Research.* (2, 1955)

GOLDRING, SIDNEY, M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Asst. Prof. of Neurol. Surgery.* (1, 1958)

GOLDRING, WILLIAM, M.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (1, 1939)

GOLDSMITH, E. D., Ph.D. New York U., New York City 10. *Prof. Histol., Grad. Sch. Arts & Sci.; Prof. Histol. & Res. Coord., Coll. Dent.* (1, 1950)

GOLDSMITH, GRACE A., M.D. Tulane U. Sch. of Med., New Orleans, La. *Prof. of Med.* (5, 1943)

GOLDSTEIN, AVRAM, M.D. Stanford U., Dept. of Pharmacol., Stanford, Calif. *Prof. and Exec. Head of Dept.* (3, 1948)

GOLDSTEIN, M. S., M.D. Michael Reese Hosp., Chigaco 16, Ill. *Assoc. Dir., Dept. of Metab. and Endocrine Res.; Lect., U. of Chicago.* (1, 1954)

GOLDSWORTHY, P. D., Ph.D. U. of Washington Sch. of Med., Depts. of Med. & Biochem., Seattle 5. *Res. Asst. Prof. of Med.; Lect. in Biochem.* (2, 1958)

GOLDTHWAIT, DAVID A., M.D. Western Reserve U., Dept. of Biochem., Cleveland 6, Ohio. *Asst. Prof.* (2, 1957)

GOLDWASSER, EUGENE, Ph.D. U. of Chicago, Argonne Cancer Research Hosp., Chicago 37, Ill. *Research Assoc.* (2, 1956)

GOLLAN, FRANK, M.D. VA Hosp., Nashville, Tenn. *Dir., Research.* (1, 1950)

GOLUB, ORVILLE JOSEPH, Ph.D. Bio-Science Labs., 12330 Santa Monica Blvd., Los Angeles 25, Calif. *Vice Pres.* (6, 1944)

GOOD, ROBERT A., M.D. U. of Minnesota, Dept. of Pediatrics, Minneapolis 14. *Amer. Legion Mem. Heart Res. Prof.* (4, 1956; 6, 1957)

GOODALE, WALTER T., M.D. 300 Longwood Ave., Boston 15, Mass. *Clin. Assoc., Harvard Med. Sch.; Sr. Assoc., P. B. Brigham Hosp.* (1, 1949)

GOODALL, McCHESNEY, M.D., Ph.D. Duke U. Med. Sch., Durham, N. C. *Prof. Exptl. Surgery; Estab. Invest., Amer. Heart Assn.* (1, 1956)

GOODMAN, HOWARD C., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Surgeon (R), PHS.* (1, 1957)

GOODMAN, IRVING, Ph.D. Wellcome Research Labs., Tuckahoe 7, N. Y. *Sr. Research Chemist.* (2, 1954)

GOODMAN, LOUIS SANFORD, M.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City. *Prof. and Chmn. of Dept.* (1, 1946; 3, 1937)

GOODMAN, MORRIS, Ph.D. Detroit Inst. of Cancer Research, Detroit 1, Mich. *Research Assoc.* (6, 1953)

GOODNER, KENNETH, Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Prof. of Bacteriol. and Immunol.* (6, 1932)

GOODPASTURE, ERNEST WILLIAM, M.D. Armed Forces Inst. of Pathol., Washington 25, D. C. *Sci. Dir. of Pathol.* (4, 1923)

GORANSON, EWALD S., Ph.D. Ontario Cancer Inst., Toronto, Canada. *Sr. Invest.* (1, 1952)

GORDON, ALBERT S., Ph.D. New York U. Grad. Sch. of Arts and Sci., New York City 3. *Prof. of Biol.* (1, 1942)

GORDON, ARCHER S., M.D., Ph.D. Presbyterian Hosp., Chicago, Ill. *Asst. Prof., Surgery, U. of Illinois Coll. of Med.* (1, 1953)

GORDON, DAVID B., Ph.D. 6465 S.W. 31st St., Miami, Fla. *Asst. Prof. of Physiol., U. of Miami Sch. of Med.* (1, 1958)

GORDON, FRANCIS B., Ph.D., M.D. Naval Med. Research Inst., Bethesda 14, Md. *Supervising Bacteriologist.* (4, 1947)

GORDON, HARRY H., M.D. Sinai Hosp. of Baltimore, Baltimore 5, Md. *Pediatrician-in-Chief.* (5, 1940)

GORDON, IRVING, M.D. U. of Southern California Sch. of Med., Dept. of Med. Microbiol., Los Angeles 7. *Prof. & Chmn. of Dept.* (6, 1943)

GORDON, WILLIAM G., Ph.D. USDA, Eastern Regional Research Lab., Philadelphia 18, Pa. *Head, Protein Structure Unit.* (2, 1939)

GORHAM, JOHN R., Ph.D. Washington State Coll., Pullman. *Veterinarian, Animal Disease and Parasite Res., USDA, ARS.* (4, 1953; 6, 1952)

GORINI, LUIGI, Ph.D. Harvard Med. Sch., Dept. of Bacteriol., Boston 15, Mass. *Lect.* (2, 1958)

GORTNER, ROSS AIKEN, JR., Ph.D. Wesleyan U., Shanklin Lab., Middletown, Conn. *Prof. of Biochem.* (5, 1947)

GORTNER, WILLIS A., Ph.D. Pineapple Research Inst. of Hawaii, Honolulu, T. H. *Head of Dept. of Chem.* (2, 1947)

GOSS, HAROLD, Ph.D. U. of California Coll. of Agric., Davis. *Prof. of Animal Husbandry.* (2, 1936; 5, 1933)

GOSSELIN, ROBERT E., Ph.D., M.D. Dartmouth Med. Sch., Dept. of Pharmacol., Hanover, N. H. *Prof. and Chmn. of Dept.* (1, 1955; 3, 1957)

GOTH, ANDRES, M.D. U. of Texas, Southwestern Med. Coll., Dept. of Pharmacol., Dallas 19. *Prof. and Head of Dept.* (3, 1947)

GOTTSCHALK, CARL W., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Asst. Prof. of Med.* (1, 1956)

GOTTSHALL, RUSSELL Y., Ph.D. Michigan Dept. of Health, Div. of Labs., Lansing. *Bacteriologist.* (6, 1939)

GOUDSMIT, ARNOLDUS, M.D., Ph.D. 2218 Market St., Youngstown 7, Ohio. (1, 1940)

GOULD, BERNARD SIDNEY, Ph.D. Massachusetts Inst. of Tech., Div. of Biochem., Cambridge. *Assoc. Prof.* (2, 1956)

GOULD, R. GORDON, Ph.D. Los Alamos Sci. Lab., Los Alamos, N. Mex. *Staff Member, Health Div.* (2, 1949)

GOURLEY, DESMOND R. H., Ph.D. U. of Virginia Med. Sch., Dept. of Pharmacol., Charlottesville. *Assoc. Prof.* (1, 1951; 3, 1954)

GOVIER, WILLIAM M., M.D. Schering Corp., Research Div., Bloomfield, N. J. *Dir. of Pharmacol.* (3, 1944)

GOWDEY, CHARLES W., Ph.D. U. of Western Ontario Med. Sch., Dept. of Pharmacol., London, Canada. *Assoc. Prof.* (3, 1950)

GRABAR, PIERRE, Ph.D., D.Sc. Institut Pasteur, Paris, France. *Chef du Service de Chimie*

Microbienne. (6, 1957)

GRABFIELD, G. PHILIP, M.D. 15 Hutchinson St., Milton 87, Mass. (3R, 1923)

GRADY, HUGH G., M.D. Seton Hall Coll. of Med. & Dent., Jersey City 4, N. J. *Prof. of Pathol.* (4, 1940)

GRAEF, IRVING, M.D. New York U., Post-Grad. Med. Sch., New York City 16. *Assoc. Prof. of Clin. Med.* (4, 1941)

GRAFF, SAMUEL, Ph.D. 211 Ft. Washington Ave., New York City 32. *Prof. of Biochem., Columbia U.* (2, 1958)

GRAFFLIN, ALLAN L., M.D. Henry Ford Hosp., Detroit 2, Mich. *Resident in Ophthalmol.* (1, 1954)

GRAHAM, CLAIRE E., Ph.D. Wilson Labs., 4221 S. Western Ave., Chicago, Ill. *Dir. of Research.* (2, 1948; 5, 1951)

GRAHAM, CLARENCE H., Ph.D. Columbia U., New York City 27. *Prof. of Psychol.* (1, 1933)

GRAHAM, HELEN TREDWAY, Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Pharmacol.* (1, 1933; 3, 1931)

GRAHAM, JOHN BORDEN. U. of North Carolina, Chapel Hill. *Prof. of Pathol.* (4, 1952)

GRAHAM, W. DONALD, Ph.D. Midwest Med. Research Fndn., 3241 Victor, Wichita 8, Kans. *Dir. of Research.* (3, 1953)

GRANDE, FRANCISCO, M.D. U. of Minnesota, Lab. of Physiol. Hygiene, Stadium Gate 27, Minneapolis 14. *Assoc. Prof.* (1, 1957; 5, 1958)

GRANICK, S., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (2, 1949)

GRANT, R. LORIMER, Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D. C. *Chief, Insulin Br.* (2, 1938)

GRANT, RHODA, Ph.D. U. of Illinois Med. Coll., Clin. Sci. Dept., Chicago 12, Ill. *Research Assoc.* (1, 1949)

GRANT, RONALD, Ph.D. Stanford U., Dept. of Physiol., Stanford, Calif. *Prof.* (1, 1950)

GRANT, WILSON CLARK, Ph.D. VA Hosp., Research Dept., Coral Gables 34, Fla. (1, 1949)

GRAU, CHARLES R., Ph.D. U. of California, Dept. of Poultry Husbandry, Davis. *Assoc. Prof.; Assoc. Nutritionist, Expt. Sta.* (5, 1949)

GRAUER, ROBERT C., M.D. Allegheny Gen. Hosp., Pittsburgh, Pa. *Dir. of William H. Singer Mem. Research Lab.* (4, 1941)

GRAY, EDWARD LeB., Ph.D. U. of Rochester Sch. of Med. and Dent., Dept. of Pharmacol. and Toxicol., Rochester 20, N. Y. *Assoc.* (1, 1951)

GRAY, GRACE W., Ph.D. Marquette U. Sch. of Med., Dept. of Pharmacol., Milwaukee 3, Wis. *Asst. Prof.* (3, 1956)

GRAY, JOHN S., Ph.D., M.D. Northwestern U. Med. Sch., Dept. of Physiol., Chicago 11, Ill. *Prof. and Chmn. of Dept.* (1, 1937)

GRAY, M. GENEVA, Ph.D. QM Research & Engin. Command, Pioneering Research Div., Natick, Mass. *Physiologist.* (3, 1946)

GRAY, STEPHEN W., Ph.D. Emory U. Sch. of Med., Atlanta 22, Ga. *Assoc. Prof. of Anatomy.* (1, 1948)

GRAY, WILLIAM D., Ph.D. American Cyanamid Co., Exptl. Therap. Res. Sec., Pearl River, N. Y. *Head, Dept. of Exptl. Pharmacol.* (3, 1955)

GRAYBIEL, ASHTON, M.D. U. S. Naval Sch. of Aviation Med., Pensacola NAS, Fla. *Capt., MC, USN; Dir. of Research.* (1, 1948)

GREELEY, PAUL O., Ph.D., M.D. U. of Southern California Sch. of Med., Los Angeles 7. *Dir., Student Health Serv.* (1, 1940)

GREEN, DANIEL M., M.D. 8877 Ladue Rd., St. Louis 24, Mo. *Vice Pres., Grove Labs., Inc.* (1, 1948; 3, 1942)

GREEN, DAVID E., Ph.D. U. of Wisconsin, Inst. for Enzyme Research, Madison 5. *Prof. of Enzyme Chem.* (2, 1941)

GREEN, HAROLD D., M.D., D.Sc. Bowman Gray Sch. of Med., Dept. of Physiol. & Pharmacol., Winston-Salem 7, N.C. *Prof. & Dir. of Dept.* (1, 1936; 3, 1945)

GREEN, HARRY, Ph.D. Wills Eye Hosp., Dept. of Research, Philadelphia 30, Pa. *Chief, Biochem. Research.* (2, 1956)

GREEN, JACK PETER, Ph.D., M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Asst. Prof.* (3, 1958)

GREEN, JOHN D., M.D. U. of California at Los Angeles, Dept. of Anatomy, Los Angeles 24. *Prof.* (1, 1954)

GREEN, RAY E., M.D. U. of Kansas Med. Ctr., Kansas City 12. *Assoc. Prof. of Pharmacol.* (3, 1952)

GREEN, ROBERT HOLT, M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Assoc. Prof. of Prev. Med.* (6, 1949)

GREENBERG, DAVID MORRIS, Ph.D. U. of California Sch. of Med., Dept. of Biochem., San Francisco 22. *Prof. and Chmn. of Dept.* (2, 1927)

GREENBERG, G. ROBERT, Ph.D. U. of Michigan Med. Sch., Dept. of Biol. Chem., Ann Arbor. *Prof.* (2, 1952)

GREENBERG, JOSEPH, M.D. Long Island Jewish Hosp., Dept. of Nuclear Med., New Hyde Park, N. Y. *Chief.* (1, 1957)

GREENBERG, LOUIS, Ph.D. Dept. of Natl. Health and Welfare, Lab. of Hygiene, Ottawa, Canada. *Chief, Biologics Control Labs.* (6, 1953)

GREENBERG, LOUIS D., Ph.D. U. of California Med. Ctr., San Francisco 22. *Assoc. Prof. of Exptl. Pathol.* (2, 1946)

GREENBERG, RUVEN, Ph.D. U. of Illinois Coll. of Med., Dept. of Physiol., Chicago. *Assoc. Prof.* (1, 1950)

GREENBERG, SAMUEL M., Ph.D. Smith Kline & French Labs., Biochem. Sec., Philadelphia 1, Pa. *Sr. Research Scientist.* (5, 1954)

GREENE, CARL H., Ph.D., M.D. 401 Clinton Ave., Brooklyn 38, N. Y. *Assoc. Prof. Clin. Med., N.Y.U. Post-Grad. Med. Sch.* (1, 1921; 2, 1922; 4, 1924)

GREENE, HARRY S. N., M.D., C.M. Yale U. Sch. of Med., Dept. of Pathol., New Haven 11, Conn. *Anthony N. Brady Prof. Pathol.* (4, 1937)

GREENE, JAMES ALEXANDER, M.D. Baylor U. Coll. of Med., Houston, Tex. *Prof. Medicine Faculty.* (1, 1939)

GREENGARD, HARRY, Ph.D., M.D. 1150 No. State St., Chicago 10, Ill. (1, 1939)

GREENSTEIN, JESSE P., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief Biochemist.* (2, 1935)

GREENWALD, ISIDOR, Ph.D. New York U. Coll. of

Med., New York City 16. *Prof. Emer., Biol. Chem.* (2R, 1912; 5R, 1933)

GREENWOOD, DELBERT A., Ph.D. Utah State U., Box 192, Logan. *Prof. of Biochem. and Pharmacol.* (3, 1950)

GREEP, ROY O., Ph.D. Harvard Sch. of Dental Med., Boston 15, Mass. *Dean and Prof. of Anatomy; Acting Head of Dept., Harvard Med. Sch.* (1, 1940)

GREER, C. M., M.S. Vanderbilt U., Nashville 5, Tenn. *Instr. in Chem.* (3, 1938)

GREGERSEN, MAGNUS I., Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Dalton Prof. of Physiol.* (1, 1933)

GREGG, DONALD E., Ph.D., M.D. Walter Reed Army Inst. of Research, Washington 12, D. C. *Chief, Dept. of Cardioresp. Diseases.* (1, 1933)

GREGORY, JOHN DELAFIELD, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (2, 1954)

GREGORY, JOHN E., M.D. Rowan Mem. Hosp., Salisbury, N. C. *Pathologist.* (6, 1948)

GREGORY, RAYMOND L., Ph.D., M.D. U. of Texas Sch. of Med., Galveston. *Prof. of Med.* (1, 1945)

GREIF, ROGER L., M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Physiol.* (1, 1956)

GREIG, MARGARET E., Ph.D. The Upjohn Co., Research Div., Kalamazoo 99, Mich. *Sr. Research Scientist.* (3, 1946)

GREINER, THEODORE H., M.D. Baylor U. Coll. of Med., Houston 25, Tex. *Asst. Prof. of Pharmacol.* (3, 1951)

GREISHEIMER, ESTHER M., Ph.D., M.D. Temple U. Hosp., Philadelphia 40, Pa. *Research Prof. of Anesthesiol.* (1, 1925)

GRENELL, ROBERT G., Ph.D. U. of Maryland Sch. of Med., Psychiatric Inst., Baltimore 1. *Prof. of Psychiatric Research.* (1, 1945)

GRIFFIN, A. CLARK, Ph.D. U. of Texas, M. D. Anderson Hosp., Dept. of Biochem., Houston. *Dept. Head.* (2, 1950)

GRIFFIN, ANGUS, Ph.D. George Washington U. Sch. of Med., Washington 5, D. C. *Prof. of Bacteriol.* (6, 1940)

GRIFFIN, DONALD R., Ph.D. Harvard U., Dept. of Biol., Cambridge 38, Mass. *Prof.* (1, 1951)

GRIFFITH, FRED R., JR., Ph.D. U. of Buffalo Sch. of Med., Dept. of Physiol., Buffalo, N. Y. *Prof. Emer.* (1R, 1923; 5R, 1933)

GRIFFITH, WENDELL H., Ph.D. U. of California Med. Ctr., Dept. of Physiol. Chem., Los Angeles 24. *Chmn. of Dept.* (2, 1923; 5, 1934)

GRIMSON, KEITH S., M.D. Duke U. Sch. of Med., Durham, N. C. *Prof. of Surgery.* (1, 1943; 3, 1949)

GRINDLAY, JOHN H., M.D. Mayo Clinic, Rochester, Minn. (1, 1945)

GRISOLIA, SANTIAGO, M.D. U. of Kansas Med. Ctr., Kansas City 3. *Assoc. Prof. of Med. and Biochem.* (2, 1952)

GROAT, RICHARD A., Ph.D., M.D. 519 Arbor Rd., Winston-Salem, N. C. (1, 1945)

GROB, DAVID, M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Med.* (1, 1951; 3, 1956)

GRODINS, FRED S., Ph.D., M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Prof. of Physiol.* (1, 1945)

GROLLMAN, ARTHUR, M.D., Ph.D. U. of Texas Southwestern Med. Sch., Dallas 19. *Prof. of Exptl. Med. and Pharmacol.* (1, 1928; 3, 1933)

GROSCHKE, ALBERT C., Ph.D. The Borden Co., 350 Madison Ave., New York City. *Tech. Dir., Feed Supplements Dept.* (5, 1952)

GROSS, ERWIN G., Ph.D., M.D. State U. of Iowa Coll. of Med., Iowa City. *Prof. of Pharmacol.* (1, 1927; 2, 1923; 3, 1927)

GROSS, J., M.D., C.M., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. (1, 1950)

GROSS, JEROME, M.D. Massachusetts Gen. Hosp., Dept. Med., Boston 14. *Assoc. Biologist; Asst. Prof. Med., Harvard Med. Sch.* (1, 1955)

GROSS, PAUL, M.D. Mellon Inst., Industrial Hygiene Fndn., Pittsburgh 13, Pa. *Research Pathologist.* (4, 1955)

GROSS, PAUL RANDOLPH, Ph.D. New York U., New York City 53. *Assoc. Prof. of Biol., Grad. Sch. and U. Coll. of Arts and Sci.* (1, 1957)

GROSS, ROBERT E., M.D. Children's Hosp., Boston 15, Mass. *Surgeon-in-Chief; Ladd Prof. of Children's Surg., Harvard Med. Sch.* (4, 1940)

GROSSMAN, JACOB, M.D. Montefiore Hosp., Med. Div., New York City 67. *Research Assoc.* (1, 1956)

GROSSMAN, MORTON IRVIN, Ph.D., M.D. VA Ctr., Los Angeles 25, Calif. *Chief, Sec. on Gastroenterol.* (1, 1946)

GROUPE, VINCENT, Ph.D. Rutgers The State U., Inst. of Microbiol., New Brunswick, N. J. *Member; Prof. of Virol.* (6, 1946)

GRUBBS, ROBERT C., M.D. Ohio State U., Columbus. *Prof. and Vice Chmn. of Physiol.* (1, 1950)

GRUBER, CHARLES M., M.D., Ph.D. 1313 College Ave., Redlands, Calif. *Emer. Prof. of Pharmacol., Jefferson Med. Coll.* (1R, 1914; 3R, 1919)

GRUBER, CHARLES M., JR., M.D., D.Sc. Lilly Lab. for Clin. Research, Indianapolis Gen. Hosp., Indianapolis 7, Ind. (3, 1948)

GRUHZIT, CARL C., M.D., Ph.D. U. of Hong Kong, Dept. of Physiol., Hong Kong. (3, 1954)

GRUHZIT, OSWALD M., M.D. 1100 Berkshire Rd., Grosse Pointe 30, Mich. *Research in Pathol. and Pharmacol.* (4, 1928)

GRUMBACH, LEONARD, Ph.D. Sterling-Winthrop Research Inst., Pharmacol. Sec., Rensselaer, N. Y. *Assoc. Member and Group Leader.* (1, 1957)

GRUNDFEST, HARRY, Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Assoc. Prof. of Neurol.* (1, 1932)

GUBLER, CLARK J., Ph.D. 1914 Rowley Ave., Madison, Wis. (5, 1951)

GUDERNATSCH, FREDRICK, Ph.D., Sc.D. Cornell U. Med. Coll., Arctic Aeromed. Bibliographic Serv., New York City 21. (1, 1930)

GUERRA, FRANCISCO, M.D. Univ. Nacional de Mexico, Fac. de Medicina, Mexico, D. F. *Prof. Titular de Carrera en Farmacologia.* (3, 1947)

GUERRANT, N. B., Ph.D. Pennsylvania State U., University Park. *Prof. of Biochem.* (2, 1934; 5, 1933)

GUEST, GEORGE MARTIN, M.D. Children's Hosp., Cincinnati 29, Ohio. *Prof. of Research Pediatrics, U. of Cincinnati Coll. of Med.* (2, 1933)

GUEST, M. MASON, Ph.D. U. of Texas, Med. Br., Dept. of Physiol., Galveston. *Prof. and Chmn. of Dept.* (1, 1946)

GUILLEMIN, ROGER, M.D., Ph.D. Baylor U. Coll. of Med., Houston 25, Tex. *Assoc. Prof. of Physiology.* (1, 1957)

GUILLEMIN, VICTOR, JR., Ph.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Biophysics.* (1, 1952)

GULICK, ADDISON, Ph.D. 3 Concord Ave., Suite 62, Cambridge 38, Mass. (1R, 1915; 5R, 1933)

GULLINO, PIETRO M., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Visiting Scientist.* (4, 1958)

GUNN, FRANCIS D., M.D., Ph.D. Latter Day Saints Hosp., Salt Lake City, Utah. *Pathologist; Clin. Prof. Pathol., U. of Utah.* (4, 1938)

GUNSALUS, IRWIN C., Ph.D. U. of Illinois, Biochem. Div., Noyes Lab., Urbana. *Prof. of Biochem.* (2, 1946)

GURD, FRANK R. N., Ph.D. Equitable Life Assurance Soc., New York City 1. *Asst. Dir. Med. Res.; Asst. Prof. Clin. Biochem., Cornell.* (2, 1952)

GURIN, SAMUEL, Ph.D. U. of Pennsylvania Sch. of Med., Dept. of Biochem., Philadelphia 4. *Prof. and Chmn.* (2, 1938)

GUSTUS, EDWIN L., Ph.D. Natl. Research Council, QM R & D Committee, Chicago, Ill. *Chmn., Committee on Foot Protection.* (2, 1934)

GUTHRIE, CHARLES C., M.D., Ph.D. 806 Hill Crest Ave., Columbia, Mo. *Prof. Emer. of Physiol. & Pharmacol., U. of Pittsburgh.* (1R, 1905; 3R, 1909)

GUTMAN, ALEXANDER B., M.D. Mt. Sinai Hosp., New York City 29. *Med. Dir.; Prof. of Med., Columbia U.* (2, 1947)

GUTMANN, H. R., M.D. VA Hosp., Minneapolis 17, Minn. *Biochemist; Assoc. Prof., Dept. of Physiol. Chem., U. of Minnesota.* (2, 1952)

GUTTMAN, RITA M., Ph.D. Brooklyn Coll., Brooklyn 3, N.Y. *Asst. Prof. of Biol.* (1, 1946)

GUYTON, ARTHUR C., M.D. U. of Mississippi Sch. of Med., Dept. of Physiol. & Biophysics, Jackson. *Prof. and Chmn. of Dept.* (1, 1949)

GYERMEK, LASZLO, M.D., C.M.Sc. U. of Illinois Coll. of Med., Chicago 12. *Asst. Prof. of Pharmacol.* (3, 1958)

GYORGY, PAUL, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Pediatrics.* (2, 1938; 5, 1939)

HAAG, HARVEY B., M.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Pharmacol.* (3, 1934)

HAAG, J. R., Ph.D. Oregon State Coll., Agric. Expt. Sta., Corvallis. *Chemist.* (2, 1947; 5, 1941)

HAAS, ERWIN, Ph.D. Mt. Sinai Hosp., 1800 E. 105th St., Cleveland 6, Ohio. *Sr. Research Chemist.* (2, 1946)

HABEL, KARL, M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Med. Dir.* (6, 1952)

HABERMAN, SOL, Ph.D. Baylor U. Hosp., Baylor U. Coll. of Dent., Dallas, Tex. *Prof. of Bacteriol. and Immunol.* (6, 1944)

HABERMANN, ROBERT T., D.V.M. Div. of Research Serv., NIH, Bethesda 14, Md. *Veterinarian Dir.* (4, 1956)

HACK, MARVIN H., Ph.D. Tulane U. Sch. of Med., Dept. of Med., New Orleans, La. *Assoc. Prof.* (2, 1954)

HACKEL, DONALD B. City Hosp., Cleveland 9, Ohio. *Assoc. Prof. of Pathol., Western Reserve U.* (1, 1955; 4, 1953)

HADDY, F. J., M.D., Ph.D. VA Research Hosp., Chicago 11, Ill. *Asst. Prof., Northwestern U., Depts. of Physiol. & Med.* (1, 1954)

HADIDIAN, ZAREH, Ph.D. Tufts U. Sch. of Med., Boston 11, Mass. *Assoc. Prof. of Pharmacol.* (1, 1945; 3, 1958)

HAFKENSCHIEL, J. H., JR., M.D. U. of Pennsylvania, Philadelphia 4. *Assoc. in Med.; Prin. Invest., Cardiopul. Unit, Lankenau Hosp.* (1, 1950; 3, 1952)

HAFKESBRING, H. ROBERTA, Ph.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. *Prof. of Physiol.* (1, 1931)

HAGANS, JAMES A., M.D. U. of Oklahoma Sch. of Med., Biostatistical Unit, Oklahoma City 4. *Instr.* (3, 1958)

HAGEN, PAUL B., M.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Pharmacol.* (3, 1956)

HAGER, LOWELL P., Ph.D. Harvard U., Dept. of Chem., Cambridge 38, Mass. *Asst. Prof.* (2, 1958)

HAGERMAN, DWAIN D., M.D. Harvard Med. Sch., 25 Shattuck St., Boston 15, Mass. *Assoc. in Biol. Chem.* (2, 1958)

HAGGARD, HOWARD W., M.D. P.O. Box 191, Tavernier, Fla. (1R, 1919; 2, 1920)

HAHN, FRED E., Ph.D. Walter Reed Army Inst. of Res., Dept. of Rickettsial Diseases, Washington 12, D.C. *Asst. Chief.* (2, 1958)

HAHN, PAUL F., Ph.D. Meharry Med. Coll., Nashville, Tenn. *Dir. of Cancer Research Labs.; Prof. of Exptl. Oncol.* (1, 1946; 4, 1939)

HAIG, CHARLES, Ph.D. New York Med. Coll., Dept. of Physiol. and Pharmacol., New York City 29. *Prof. of Physiol.* (1, 1942)

HAIMOVICI, HENRY, M.D. 105 E. 90th St., New York City 28. *Attending Vascular Surgeon, Montefiore Hosp.* (1, 1950)

HAINES, WILLIAM J., Ph.D. Childsworth Ave., Basking Ridge, N.J. *Vice Pres. & Dir. of Research, Ortho Pharmaceutical Corp., Raritan.* (2, 1953)

HAIST, REGINALD E., M.D., Ph.D. U. of Toronto, Toronto, Canada. *Prof. of Physiol.* (1, 1943)

HAJEK-NICHOLS, NORMA M., Ph.D. Med. Coll. of Virginia, Dept. of Physiol., Richmond 19. *Asst. Prof.* (1, 1954)

HALBERG, FRANZ, M.D. U. of Minnesota Med. Sch., Dept. Pathol., Minneapolis 14. *Prof.; Dir. of Res., Cambridge State Hosp.* (1, 1955)

HALBERT, SEYMOUR P., M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Microbiol.* (6, 1947)

HALDI, JOHN, Ph.D. Emory U., Dept. of Physiol., Atlanta 22, Ga. *Prof.* (1, 1928)

HALE, WILLIAM M., M.D. U. of Tennessee Sch. of Med., Cancer Research Lab., Memphis. *Prof. of Bacteriol.* (4, 1941; 6, 1935)

HALE, WORTH, M.D. Silver St., Monson, Mass. (1R, 1908; 3R, 1908)

HALEY, THOMAS J., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Chief, Div. of Pharmacol. & Toxicol., A. E. Project.* (3, 1949)

HALL, CHARLES E., Ph.D. U. of Texas Med. Br.,

Dept. of Physiol., Galveston. *Prof.* (1, 1949)

Hall, F. G., Ph.D. Duke U. Sch. of Med., Dept. of Physiol. & Pharmacol., Durham, N. C. *Prof. of Physiol.; Chmn. of Dept.* (1, 1937)

HALL, JOHN F., JR., M.A. 368 Wendell Lane, Dayton 3, Ohio. *Research Physiologist, USAF Aero-Med. Lab., Wright-Patterson AFB.* (1, 1957)

HALL, ROSS H., Ph.D. Amer. Cyanamid Co., Research Div., Pearl River, N. Y. *Research Chemist.* (2, 1958)

HALL, VICTOR E., M.D. 506 Hanley Pl., Los Angeles 49, Calif. *Prof. of Physiol., U. of California Sch. of Med.* (1, 1934)

HALL, W. KNOWLTON, Ph.D. Med. Coll. of Georgia, Augusta. *Prof. of Biochem.* (2, 1948)

HALLENBECK, GEORGE AARON, Ph.D., M.D. Mayo Clinic, Rochester, Minn. *Assoc. Prof. of Physiol. and Surgery.* (1, 1946)

HALPERT, BELA, M.D. VA Hosp., Houston 31, Tex. *Chief, Lab. Serv.; Prof. of Pathol., Baylor U. Coll. of Med.* (4, 1936)

HALSTEAD, WARD C., Ph.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Prof. of Exptl. Psychol.; Dir. of Med. Psychol.* (1, 1942)

HALSTED, JAMES A., M.D. VA Hosp., Syracuse, N. Y. *Dir., Prof. Serv.; Assoc. Prof. Dept. Med., State U. of New York Coll. Med.* (4, 1956)

HALVER, JOHN E., Ph.D. Underwood, Wash. *Chief, Western Fish Nutrition Invest., Cook, Wash.* (5, 1958)

HAM, ARTHUR W., M.B. Ontario Cancer Inst., Div. Biol. Res., Toronto 5, Canada. *Head; Prof. & Head, Med. Biophysics, U. Toronto.* (4, 1939)

HAMBOURGER, WALTER E., Ph.D., M.D. G. D. Searle & Co., P. O. Box 5110, Chicago 80, Ill. *Chief Pharmacologist.* (3, 1934)

HAMILTON, BENGT L. K., M.D. 8311 Drexel Ave., Chicago 19, Ill. *Med. Dir., PHS (Retired).* (2, 1925)

HAMILTON, CLARA E., Ph.D. Div. of Research Grants, NIH, Bethesda 14, Md. (1, 1952)

HAMILTON, JAMES B., Ph.D. State U. of New York Coll. of Med., Dept. of Anatomy, Brooklyn 3. *Prof.* (1, 1938)

HAMILTON, JOHN D., M.D. U. of Toronto, Banting Inst., Dept. of Pathol., Toronto 2, Canada. *Prof. and Head of Dept.* (4, 1957)

HAMILTON, LEONARD D., Ph.D. Sloan-Kettering Inst. for Cancer Res., New York City 21. *Head, Isotope Studies Sec.* (4, 1958)

HAMILTON, LYLE H., Ph.D. Wood VA Hosp., Med. Research Serv., Wood, Wis. *Chief, Physiol. Sec.* (1, 1957)

HAMILTON, PAUL B., M.D., Ph.D. Alfred I. duPont Inst., Nemours Fndn., Rockland Rd., Wilmington 99, Del. *Chief of Biochem.* (2, 1946)

HAMILTON, TOM R., M.D. Kansas U. Med. Ctr., Kansas City 3. *Prof. of Microbiol. and Pathol.; Chmn., Med. Microbiol.* (4, 1950)

HAMILTON, TOM S., Ph.D. U. of Illinois, 107 Mumford Hall, Urbana. *Prof. Animal Nutr.; Assoc. Dir., Ill. Agric. Expt. Sta.* (2, 1937; 5, 1938)

HAMILTON, W. F., Ph.D. Med. Coll. of Georgia, Augusta. *Prof. of Physiol.* (1, 1924)

HAMMON, W. McD., M.D., D.P.H. U. of Pittsburgh, Pittsburgh 13, Pa. *Prof. Epidemiol.; Head, Dept. Epidemiol. & Microbiol., Grad. Sch.* (4, 1944; 6, 1946)

HAMMOND, CAROLYN W., Ph.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Research Assoc.* (4, 1954)

HAMPEL, C. W., Ph.D. New York U. Coll. of Dent., New York City 10. *Prof. of Physiol. and Biophysics.* (1, 1936)

HAMPTON, JOHN K., JR., Ph.D. Tulane U. Sch. of Med., Dept. of Physiol., New Orleans 18, La. *Assoc. Prof.* (1, 1954)

HAMRE, DOROTHY, Ph.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Research Assoc.* (6, 1948)

HANAHAN, DONALD J., Ph.D. U. of Washington, Dept. of Biochem., Seattle 5. *Assoc. Prof.* (2, 1952)

HAND, DAVID B., Ph.D. New York State Agric. Expt. Sta., Cornell U., Geneva. *Head, Dept. of Food Sci. and Tech.; Prof. of Biochem.* (2, 1947)

HANDFORD, STANLEY WING, Ph.D. Naval Med. Research Inst., Bethesda 14, Md. (1, 1954)

HANDLER, ALFRED H., Ph.D. Children's Med. Ctr., Boston, Mass. *Research Assoc.* (4, 1957)

HANDLER, PHILIP, Ph.D. Duke U. Sch. of Med., Durham, N. C. *Prof. of Biochem. and Nutrition.* (2, 1944; 5, 1946)

HANDLEY, CARROLL A., Ph.D. Baylor U. Coll. of Med., Dept. of Pharmacol., Houston 25, Tex. *Prof. and Chmn. of Dept.* (3, 1942)

HANES, CHARLES S., Ph.D. U. of Toronto, Toronto, Canada. *Prof. of Biochem.* (2, 1954)

HANEY, HANCE F., Ph.D., M.D. U. of Oregon Med. Sch., Portland 5. *Prof. of Med.* (1, 1939)

HANGER, FRANKLIN M., M.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Med.* (6, 1930)

HANKE, MARTIN E., Ph.D. U. of Chicago, Chicago 37, Ill. *Assoc. Prof. of Biochem.* (2, 1925)

HANKE, MILTON THEO, Ph.D. 3553 S. Indian River Dr., Fort Pierce, Fla. (2, 1919)

HANKES, LAWRENCE V., Ph.D. Brookhaven Natl. Lab., Upton, N. Y. *Biochemist; Head of Clin. Chem.* (2, 1955)

HANKS, JOHN H., Ph.D. Harvard U. Med. Sch., Dept. of Bacteriol., Boston 15, Mass. *Bacteriologist of Leonard Wood Mem. Fndn.* (6, 1935)

HANNA, CALVIN, Ph.D. U. of Vermont Coll. of Med., Dept. of Pharmacol., Burlington. *Assoc. Prof.* (3, 1955)

HANNON, JOHN PATRICK, Ph.D. Arctic Aeromed. Lab., Biochem. Dept., APO 731, Seattle, Wash. *Res. Physiologist.* (1, 1958)

HANSARD, SAMUEL L., Ph.D. Louisiana State U., Dept. of Animal Industry, Baton Rouge. *Animal Nutritionist.* (5, 1955)

HANSEN, ARILD E., M.D. U. of Texas Sch. of Med., Dept. of Pediatrics, Galveston. *Prof. and Chmn. of Dept.* (4, 1941; 5, 1942)

HANSEN, EDER LINDSAY, Ph.D. 561 Santa Barbara Rd., Berkeley 7, Calif. *Research Assoc. in Pharmacol.* (3, 1951)

HANSEN, ROGER G., Ph.D. Michigan State U., Dept. of Agric. Chem., East Lansing. *Prof. and Head of Dept.* (2, 1952)

HARARY, ISAAC, Ph.D. VA Res. Hosp., Radioisotope Unit, Chicago, Ill. *Asst. Prof., Northwestern U.* (2, 1958)

HARBURY, HENRY A., Ph.D. Yale U., Dept. of Biochem., New Haven, Conn. *Asst. Prof.* (2, 1958)

HARDENBERGH, ESTHER, S.D. Naval Med. Research Inst., Div. of Physiol., Bethesda 14, Md. *Head, Circulatory Studies Br.* (1, 1951)

HARDY, JAMES D., M.D. U. of Mississippi Med. Ctr., Jackson. *Prof. and Chmn., Dept. of Surgery; Surgeon-in-Chief, Univ. Hosp.* (1, 1951)

HARDY, JAMES DANIEL, Ph.D. Naval Air Develop. Ctr., Aviation Med. Lab., Johnsville, Pa. *Dir. of Res.; Prof., Physiol., U. Pa.* (1, 1939)

HARDY, MARY, D.Sc. The Chapin School, 100 East End Ave., New York City. *Teacher of Biol. and Physics.* (1, 1933)

HARE, KENDRICK, Ph.D., M.D. Med. Coll. of Alabama, Birmingham 5. *Prof. of Pediatrics and Pharmacol.* (1, 1938)

HARFORD, CARL G., M.D. Washington U. Sch. of Med., Dept. of Med., St. Louis 10, Mo. *Assoc. Prof.* (6, 1948)

HARGER, R. N., Ph.D. Indiana U. Sch. of Med., Indianapolis 7. *Prof. of Biochem. and Toxicol.* (2, 1938; 3, 1949)

HARKIN, JAMES C., M.D. Washington U. Sch. of Med., Dept. of Pathol., St. Louis 10, Mo. *Asst. Prof.* (4, 1957)

HARKINS, HENRY N., Ph.D., M.D. U. of Washington Sch. of Med., Dept. of Surgery, Seattle 5. *Prof. and Exec. Officer.* (1, 1942)

HARMAN, JOHN W., M.D. University Coll., Dept. of Pathol., Dublin, Ireland. (4, 1951)

HARMON, PAUL M., Ph.D. Indiana U., Bloomington. *Prof. of Physiol.* (1, 1930)

HARNE, O. G. 8417 Merrymount Dr., Baltimore 7, Md. (1R, 1935)

HARNED, BEN KING, Ph.D. Mead Johnson & Co., Research Lab., Evansville 21, Ind. *Vice Pres. for Research.* (2, 1931; 3, 1941)

HARPER, ALFRED E., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Assoc. Prof.* (2, 1958; 5, 1955)

HARPER, HAROLD A., Ph.D. U. of California Med. Ctr., San Francisco 22. *Assoc. Prof. of Physiol. Chem.* (2, 1955)

HARRINGTON, WILLIAM F., Ph.D. Natl. Heart Inst., Lab. of Cellular Physiol. & Metab., NIH, Bethesda 14, Md. *Biochemist.* (2, 1958)

HARRINGTON, WILLIAM J., M.D. Washington U. Sch. of Med., St. Louis, Mo. *Assoc. Prof. of Med.; Asst. Physician, Barnes Hosp.* (4, 1956)

HARRIS, ALBERT H., M.D. New York State Dept. of Health, Div. of Labs. & Research, Albany 1. *Asst. Dir.* (6, 1937)

HARRIS, ALBERT SIDNEY, Ph.D. Louisiana State U. Sch. of Med., Dept. of Physiol., New Orleans 12. *Prof. and Head of Dept.* (1, 1939)

HARRIS, CHARLES, M.D. Temple U. Med. Sch., Fels Research Inst., Philadelphia 22, Pa. *Assoc. in Pathol.* (4, 1956)

HARRIS, DANIEL L., Ph.D. U. of Chicago, Dept. of Physiol., Chicago 37, Ill. *Assoc. Prof.* (2, 1956)

HARRIS, LORIN E., Ph.D. Utah State U., Logan. *Prof. of Animal Husbandry.* (5, 1953)

HARRIS, MILTON, Ph.D. Harris Research Labs., Inc., 1246 Taylor St., N. W., Washington 11, D. C. *Pres.* (2, 1939)

HARRIS, PAUL N., M.D. Eli Lilly & Co., Div. of Pharmacol., Indianapolis 6, Ind. *Pathologist.* (3, 1948; 4, 1955)

HARRIS, PHILIP L., Ph.D. Distillation Products Ind., Rochester 3, N. Y. *Asst. Dir. Res.; Assoc. in Physiol., U. of Rochester.* (2, 1946; 5, 1945)

HARRIS, ROBERT S., Ph.D. Massachusetts Inst. of Tech., Cambridge. *Prof. of Biochem. of Nutrition.* (5, 1941)

HARRIS, S. C., Ph.D. Northwestern U. Dental Sch., Dept. of Physiol. & Pharmacol., Chicago 11, Ill. *Prof. and Chmn.* (1, 1947; 3, 1952)

HARRIS, T. N., M.D. Children's Hosp. of Philadelphia, Philadelphia, Pa. *Prof. of Immunol. in Pediatrics, U. of Pennsylvania.* (6, 1946)

HARRIS, WILLIAM H., M.D. 1215 Lowerline St., New Orleans 18, La. *Emer. Prof. of Pathol., Tulane U.* (4, 1925)

HARRISON, FRANK, Ph.D., M.D. U. of Texas South-Western Med. Sch., Dallas 19. *Prof. of Anatomy and Assoc. Dean.* (1, 1941)

HARRISON, HELEN COPLAN, Ph.D. Johns Hopkins U. Sch. of Med., Dept. of Pediatrics, Baltimore 5, Md. *Research Assoc.* (1, 1957)

HARRISON, JAMES A., Ph.D. Temple U., Philadelphia 22, Pa. *Prof. of Biol.* (6, 1946)

HARRISON, R. WENDELL, Ph.D. 5541 Dorchester Ave., Chicago 37, Ill. *Prof. of Microbiol.; Dean of Faculties; Vice-Pres., U. of Chicago.* (6, 1934)

HARRISON, ROSS GRANVILLE, M.D., Ph.D. Yale U., Osborn Zool. Lab., New Haven, Conn. *Sterling Prof. Emer. of Biol.* (1R, 1906)

HARROW, BENJAMIN, Ph.D. 333 Central Park West, New York City 25. *Prof. Emer. of Chem., CCNY.* (2R, 1927)

HART, E. ROSS, Ph.D. VA Res. Labs. in Neuropsychiatry, Pittsburgh 6, Pa. *Chief, Div. of Neurophysiol. & Neuropharmacol.* (3, 1944)

HART, J. SANFORD, Ph.D. Natl. Research Labs., Biology Div., Ottawa 2, Canada. *Sr. Research Officer.* (1, 1955)

HART, WILLIAM M., Ph.D., M.D. Georgetown U., Washington 7, D. C. *Clin. Assoc. Prof., Ophthalmol. and Spec. Lect. in Physiol.* (1, 1945)

HARTE, ROBERT A., B.S. Merck Sharp & Dohme Research Labs. Div., Merck & Co., Inc., West Point, Pa. *Exec. Asst. for Admin.* (5, 1953)

HARTLINE, H. K., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (1, 1929)

HARTMAN, ARTHUR M., B.S. USDA, Bur. of Dairy Industry, Div. of Nutrition and Physiol., Beltsville, Md. *Biochemist.* (5, 1952)

HARTMAN, CARL G., Ph.D. 219 Norwood Ave., North Plainfield, N. J. *Prof. of Zool. and Physiol. Emer., U. of Illinois.* (1R, 1921)

HARTMAN, ELIZABETH CROFTS, M.S. Natl. Inst. of Neurological Diseases and Blindness, NIH, Bethesda 14, Md. (5, 1949)

HARTMAN, F. W., M.D. 7508 Glenriddle Rd., Bethesda 14, Md. *Med. Research Advisor to Surgeon Gen., USAF.* (4, 1927)

HARTMAN, FRANK ALEXANDER, Ph.D. Ohio State U., Dept. of Physiol., Columbus. *Research Prof. Emer.* (1R, 1916)

HARTMAN, VIVIAN BEHRMANN, Ph.D. 7508 Glen-

riddle Rd., Bethesda 14, Md. *Research Physiologist.* (1, 1948)

HARTMANN, ALEXIS F., SR., M.D. 500 S. Kingshighway, St. Louis 10, Mo. *Prof. of Pediatrics, Washington U. Sch. of Med.* (2, 1932)

HARTROFT, WALTER STANLEY, M.D., Ph.D. Washington U. Med. Sch., Dept. of Pathol., St. Louis 10, Mo. *Prof. and Chmn. of Dept.* (4, 1950)

HARVEY, A. McGEHEE, M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Prof. of Med., Johns Hopkins U. Med. Sch.* (1, 1946; 3, 1946)

HARVEY, E. NEWTON, Ph.D., D.Sc. Princeton U., Guyot Hall, Princeton, N. J. *Prof. of Biol., Emer.* (1R, 1914; 2R, 1916)

HARVEY, RODNEY B., M.D., Ph.D. U. of Utah, Dept. of Physiol., Salt Lake City 12. *Asst. Prof.* (1, 1956)

HARVEY, STEWART C., Ph.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City 12. *Assoc. Prof.* (3, 1951)

HASS, GEORGE, M.D. Presbyterian-St. Luke's Hosp., Chicago, Ill. *Chmn. Rush Dept. of Pathol.; Prof. of Pathol., U. of Illinois.* (4, 1939)

HASSETT, CHARLES C., Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Chief, Entomol. Br.* (1, 1954)

HASSID, WILLIAM Z., Ph.D. U. of California, 337 Biochem.-Virus Lab., Berkeley 4. *Prof. of Plant Biochem.* (2, 1946)

HASTINGS, A. BAIRD, Ph.D. Harvard Med. Sch., Boston 15, Mass. *Hamilton Kuhn Prof. Biol. Chem.* (1, 1927; 2, 1921; 5, 1940)

HATHAWAY, MILICENT L., Ph.D. USDA, ARS, Human Nutrition Res. Div., Washington 25, D. C. *Nutrition Specialist.* (5, 1945)

HAUCK, HAZEL M., Ph.D. Cornell U. Coll. of Home Econ., Ithaca, N. Y. *Prof. of Food and Nutrition.* (5, 1941)

HAUGAARD, NIELS, Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof. of Pharmacol.* (2, 1953; 3, 1958)

HAUROWITZ, FELIX, M.D., D.Sc. Indiana U., Bloomington. *Prof. of Chem.* (2, 1949; 6, 1948)

HAUSBERGER, FRANZ X., M.D. Jefferson Med. Coll. of Philadelphia, Dept. of Anatomy, Philadelphia 7, Pa. *Assoc. Prof.* (1, 1957)

HAUSMANN, WERNER, D.Sc. American Cyanamid Co., Research Div., Pearl River, N. Y. (2, 1956)

HAVEN, FRANCES L., Ph.D. U. of Rochester Med. Ctr., Rochester 20, N. Y. *Res. Asst. Prof. of Biochem.* (2, 1941)

HAWK, PHILIP B., Ph.D. 750 W. 50th St., Miami Beach, Fla. (1, 1903; 2, 1906)

HAWKINS, J. E., JR., Ph.D. New York U.-Bellevue Med. Ctr., Dept. of Otolaryngol., New York City 16. *Assoc. Prof.* (1, 1943)

HAWKINS, W. W., Ph.D. Natl. Research Council of Canada, Atlantic Regional Lab., Halifax, Canada. *Assoc. Research Officer.* (2, 1953)

HAWKINS, WILLIAM BRUCE, M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N. Y. *Prof. of Pathol.* (4, 1933)

HAWLEY, ESTELLE E., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N. Y. *Assoc. in Pediatrics and Nutrition.* (5, 1935)

HAWN, CLINTON v. Z., M.D. Mary Imogene Bassett Hosp., Cooperstown, N. Y. *Pathologist-in-Chief; Assoc. Prof. Pathol., Columbia U.* (4, 1948)

HAWTHORNE, EDWARD W., M.D., Ph.D. Howard U. Coll. of Med., Dept. of Physiol., Washington 1, D. C. *Assoc. Prof.; Head of Dept.* (1, 1955)

HAY, ELEANOR CLARKE, Ph.D. 7 Greenhill Rd., Madison, N. J. (1, 1945)

HAYAISHI, OSAMU, M.D. Kyoto U. Faculty of Med., Dept. Med. Chem., Sakyoku, Kyoto, Japan. *Prof. and Chmn.* (2, 1952)

HAYANO, MIKA, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Research Biochemist.* (2, 1952)

HAYASHI, TERU, Ph.D. Columbia U., 1324 Pupin Hall, New York City 27. *Prof. of Zool.* (1, 1951)

HAYES, SHELDON P., Ph.D. Weber Coll., Dept. of Bacteriol., 3800 Harrison Ave., Ogden, Utah. *Head of Dept.* (6, 1953)

HAYES, WAYLAND JACKSON, JR., M.D., Ph.D. USPHS, Tech. Develop. Labs., CDC, P. O. Box 769, Savannah, Ga. *Chief, Toxicol. Sec.* (3, 1958)

HAYMAN, J. M., JR., M.D. Tufts U. Sch. of Med., Boston 11, Mass. *Dean and Prof. of Med.* (1, 1928; 3, 1932)

HAYNES, FLORENCE W., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Research Assoc. in Med.* (1, 1937)

HAYS, EDWIN EVERETT, Ph.D. Strasenburgh Labs., P. O. Box 1710, Rochester 3, N. Y. *Dir. of Research.* (2, 1946)

HAYS, HARRY W., Ph.D. Natl. Research Council, 2101 Constitution Ave., Washington 25, D. C. *Dir., Toxicol. Information Ctr.* (3, 1949)

HAYWOOD, CHARLOTTE, Ph.D. Mount Holyoke Coll., South Hadley, Mass. *Prof. of Physiol.* (1, 1939)

HAZEN, ELIZABETH L., Ph.D. New York State Dept. of Health, Div. of Labs. & Res., Albany 1. *Assoc. Bacteriologist (Mycol.).* (6, 1931)

HAZLETON, LLOYD W., Ph.D. Box 333, Falls Church, Va. *Pres., Hazleton Labs.* (3, 1944)

HEAGY, FRED CLARK, M.D., Ph.D. U. of Western Ontario, Dept. of Biochem., Faculty of Med., London, Canada. *Asst. Prof.* (2, 1954)

HEARON, JOHN Z., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Mathematician.* (1, 1951)

HECHT, HANS H., M.D. Salt Lake Gen. Hosp., Salt Lake City, Utah. *Prof. of Med., U. of Utah Coll. of Med.* (1, 1952)

HECHTER, OSCAR M., Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Member.* (1, 1945)

HEGNAUER, ALBERT H., Ph.D. Boston U. Sch. of Med., Boston, Mass. *Prof. of Physiol.* (1, 1937)

HEGSTED, DAVID MARK, Ph.D. Harvard Sch. of Public Health, 1 Shattuck St., Boston, Mass. *Assoc. Prof. of Nutrition.* (5, 1944)

HEIDELBERGER, CHARLES, Ph.D. U. of Wisconsin, McArdle Mem. Lab., Madison 6. *Assoc. Prof. of Oncol.* (2, 1950)

HEIDELBERGER, MICHAEL, Ph.D. Rutgers, The State U., Inst. of Microbiol., New Brunswick, N. J. *Visiting Prof. of Immunochem.* (2R, 1927; 6, 1935)

HEILBRUNN, LEWIS VICTOR, Ph.D. U. of Pennsylvania, Philadelphia 4. *Prof. of Zool.* (1, 1930)

HEINBECKER, PETER, M.D. Washington U. Med. Sch., St. Louis 10, Mo. *Prof. of Clin Surgery.*

(1, 1930)

HEINLE, ROBERT W., M.D. The Upjohn Co., Research Div., Kalamazoo, Mich. *Dir. of Biol. Research.* (5, 1948)

HEINRICH, MAX A., JR., Ph.D. U. of South Dakota, Dept. of Physiol. and Pharmacol., Vermillion. *Asst. Prof.* (3, 1956)

HEINRICH, MILTON R., Ph.D. U. of California, Biochem. Dept., Berkeley 4. *Research Fellow.* (2, 1958)

HEIM, J. WILLIAM, Ph.D. 2366 S. Patterson Blvd., Dayton 9, Ohio. *Chief, Physiol. Br. Aero-Med. Lab., Wright-Patterson AFB.* (1, 1936)

HEINZ, ERICH, M.D. Tufts U. Med. Sch., Dept. of Biochem. and Nutrition, Boston, Mass. *Assoc. Prof.* (2, 1955)

HELFF, O. M., Ph.D. New York U., University Heights, New York City 53. *Assoc. Prof. of Biol.* (1, 1932)

HELLBAUM, ARTHUR A., Ph.D., M.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Prof. of Pharmacol.* (1, 1937; 3, 1945)

HELLEBRANDT, FRANCES ANNA, M.D. RFD 3, Cable Lane, Athens, Ohio. (1R, 1933)

HELLER, CARL G., M.D., Ph.D. Pacific Northwest Research Fndn., Seattle 4, Wash. *Research Assoc., Endocrinol. Div.* (1, 1945)

HELLER, JOHN H., M.D. New England Inst. for Med. Research, Ridgefield, Conn. *Exec. Dir.* (1, 1955)

HELLER, VICTOR G., Ph.D. Oklahoma State U., Dept. of Biochem., Stillwater. *Emer. Prof. and Head.* (2R, 1935; 5R, 1935)

HELLERMAN, LESLIE, Ph.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Physiol. Chem.* (2, 1935)

HELLERSTEIN, EARL E., M.D. Harvard Med. Sch., Boston 15, Mass. *Research Assoc. in Pathol.* (4, 1958)

HELLERSTEIN, H. K., M.D. Western Reserve U., Cleveland 6, Ohio. *Asst. Prof. of Med.; Dir., Work Classification Clinic.* (1, 1957)

HELMER, OSCAR MARVIN, Ph.D. Lilly Lab. of Clin. Res., Indianapolis Gen. Hosp., Indianapolis 7, Ind. *Head, Dept. Clin. Biochem.* (2, 1935)

HEMING, ARTHUR E., Ph.D. Smith, Kline & French Labs., Philadelphia 1, Pa. *Assoc. Dir. of Res., Res. & Develop. Div.* (3, 1954)

HEMINGWAY, ALLAN, Ph.D. U. of California Sch. of Med., Dept. of Physiol., Los Angeles 24. *Prof.* (1, 1933)

HENDERSHOT, LELAND C., D.D.S., Ph.D. Dow Chem. Co., Biochem. Research Lab., Midland, Mich. *Pharmacologist.* (3, 1957)

HENDERSON, FRANCIS G., M.D. 740 S. Alabama St., Indianapolis 2, Ind. *Pharmacologist, Eli Lilly & Co.* (3, 1950)

HENDERSON, LaVELL M., Ph.D. Oklahoma State U., Dept. of Biochem., Stillwater. *Prof.* (2, 1949; 5, 1958)

HENDLER, RICHARD W., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Biochemist, Lab. of Cellular Physiol. and Metab.* (2, 1957)

HENDLEY, CHARLES D., Ph.D. Schering Corp., Bloomfield, N. J. *Sr. Pharmacologist.* (1, 1949)

HENDLEY, EDITH D., Ph.D. 5321 Lothian Rd., Baltimore 12, Md. (1, 1955)

HENDRIX, JAMES PAISLEY, M.D. Duke Hosp., Durham, N. C. *Assoc. Prof. of Med. and Therap.* (3, 1942)

HENDRIX, ROBERT COWGILL, M.D. U. of Michigan Med. Sch., Dept. of Pathol., Ann Arbor. *Assoc. Prof.* (4, 1954)

HENDRY, JESSIE L., M.A. New York State Dept. of Health, Div. of Labs. and Research, Albany. *Assoc. Bacteriologist.* (6, 1938)

HENLE, WERNER, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Virol.; Member, Res. Staff, Children's Hosp.* (6, 1938)

HENNEMAN, ELWOOD, M.D., C.M. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Physiol.* (1, 1952)

HENNESSY, ALBERT V., M.D. U. of Michigan Sch. of Public Health, Ann Arbor. *Asst. Prof., Epidemiol.; Asst. Prof. in Pediatrics, Med. Sch.* (6, 1955)

HENNIGAR, GORDON ROSS, JR., M.D. State U. of New York Coll. of Med., Brooklyn 3. *Prof. of Pathol.* (4, 1956)

HENRY, JAMES P., M.D., Ph.D. European Office, ARDC, Brussels, Belgium. *Lt. Col., USAF (MC); Chief, Biosciences, Tech. Op. Div.* (1, 1947)

HENSCHEL, AUSTIN F., Ph.D. QM Research & Engin. Command, Natick, Mass. *Chief, Environ. Protection Res. Div.; Dir. of Lab.* (1, 1944)

HEPBURN, J. S., Ph.D., M.D. 2045 N. Franklin, Philadelphia, Pa. *Prof. Chem.; Res. Assoc. Gastroenterol., Habnemann, Emer.* (2R, 1915)

HEPLER, OPAL E., Ph.D., M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Assoc. Prof. of Pathol. and Dir. of Labs.* (4, 1939)

HEPPEL, LEON A., Ph.D., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Surgeon, PHS.* (2, 1949)

HERBST, ROBERT M., Ph.D. Michigan State U., Kedzie Chemical Lab., East Lansing. *Prof. of Chem.* (2, 1938)

HERGET, CARL M., Ph.D. 2163 Firethorn Rd., Baltimore 20, Md. *Chief, Biophysics Div., Chem. Warfare Labs.* (1, 1948)

HERNANDEZ, THOMAS, M.D., Ph.D. Louisiana State U. Sch. of Med., Biochem. Dept., New Orleans 12. *Assoc. Prof.* (1, 1955)

HERRICK, C. JUDSON, Ph.D. 236 Morningside Dr., Grand Rapids 6, Mich. *Prof. Emer. of Neurol., U. of Chicago.* (1R, 1907)

HERRICK, JULIA F., Ph.D. Mayo Fndn., Rochester, Minn. *Prof. of Biophysics.* (1, 1933)

HERRIN, RAYMOND C., Ph.D., M.D. U. of Wisconsin Med. Sch., Madison 6. *Prof. of Physiol.* (1, 1932)

HERRINGTON, LOVIC P., Ph.D. 290 Congress Ave., New Haven, Conn. *Dir. Res., John B. Pierce Fndn.; Lect., Dept. Public Health, Yale Med. Sch.* (1, 1942)

HERRIOTT, ROGER M., Ph.D. Johns Hopkins Sch. of Hygiene & Public Health, Dept. of Biochem., Baltimore 5, Md. *Prof. and Head.* (2, 1940)

HERRMANN, GEORGE R., Ph.D., M.D. U. of Texas Med. Br., Galveston. *Prof. of Med.; Dir. of Cardiovasc. Research.* (4R, 1925)

HERRMANN, JULIAN B., Ph.B., M.D. 1185 Park Ave., New York City. (3, 1941)

HERRMANN, LOUIS GEORGE, M.D. Cincinnati Gen. Hosp., Cincinnati 29, Ohio. *Assoc. Prof. of Sur-*

gery, U. of Cincinnati Coll. of Med. (4, 1933)

HERRMANN, ROY G., Ph.D. Lilly Research Labs., Eli Lilly & Co., Indianapolis 6, Ind. *Pharmacologist.* (3, 1954)

HERSHEY, SOLOMON G., M.D. New York U., Postgrad. Med. Sch., 750 Ladd Rd., New York City 71. *Clin. Prof. of Anesthesiol.* (3, 1952)

HERTIG, ARTHUR T., M.D. Harvard Med. Sch., Boston 15, Mass. *Shattuck Prof. of Pathol. Anatomy.* (4, 1956)

HERTZ, ROY, Ph.D., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief, Endocrinol. Br.* (1, 1945)

HERTZMAN, ALRICK B., Ph.D. St. Louis U. Sch. of Med., St. Louis, Mo. *Prof. of Physiol. and Dir. of Dept.* (1, 1925)

HERWICK, ROBERT P., Ph.D., M.D. Ethicon, Inc., Somerville, N. J. *Dir. of Clin. Investigation.* (3, 1938)

HESS, CHARLES L., M.D. 110 E. Coronado Rd., Phoenix, Ariz. (1R, 1916)

HESS, EUGENE LYLE, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Sr. Scientist, Biophysical Sec.* (2, 1956)

HESS, GEORGE PAUL, Ph.D. Cornell U., Dept. of Biochem. and Nutrition, Ithaca, N. Y. *Asst. Prof. of Biochem.* (2, 1958)

HESS, WALTER C., Ph.D. Georgetown U. Med. Sch., Washington 7, D. C. *Asst. Dean and Prof. of Biol. Chem.* (2, 1935)

HESS, WALTER RUDOLF, M.D. U. of Zurich, Zurich, Switzerland. *Prof. Emer. of Physiol.* (1H, 1950)

HESTON, WALTER E., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Gen. Biol. Sec.* (4, 1953)

HETHERINGTON, ALBERT W., Ph.D. Hq, Air Research and Develop. Command, Washington 25, D. C. (1, 1943)

HEWETSON, JEAN HAWKS, Ph.D. Rt. 3, Gettysburg, Pa. (5, 1944)

HEWITT, EARL ALBON, Ph.D. Iowa State Coll., Ames. *Prof., Dept. of Veterinary Physiol. and Pharmacol.* (1, 1932)

HEWSTON, ELIZABETH M., M.A. 1121-24th St., N. W., Washington 7, D. C. *Chemist, Home Econ. Inst., ARS, USDA.* (5, 1950)

HEYMANS, C., M.D. U. of Ghent, 3 Albert Baertsoenkaai, Ghent, Belgium. *Prof. of Pharmacol.* (3, 1951)

HEYROTH, FRANCIS F., M.D., Ph.D. 110 Hosea Ave., Clifton, Cincinnati 20, Ohio. (2R, 1935)

HEYWANG, BURT W., M.S. USDA, Southwest Poultry Expt. Sta., Glendale, Ariz. (5, 1956)

HIATT, E. P., Ph.D., M.D. Aero Med. Lab., WCLDB-1, Wright Air Develop. Ctr., Wright-Patterson AFB, Ohio. *Chief, Accel. Sec.* (1, 1942)

HIATT, HOWARD H., M.D. Beth Israel Hosp., Boston 15, Mass. *Assoc. in Med., Harvard Med. Sch.* (2, 1957)

HIBBS, JOHN W., Ph.D. Ohio Agric. Expt. Sta., Dept. of Dairy Sci., Wooster. *Prof.* (5,1958)

HICKAM, JOHN B., M.D. Indiana U. Med. Ctr., Indianapolis 7. (1, 1951)

HICKS, SAMUEL P., M.D. New England Deaconess Hosp., Boston 15, Mass. *Patbologist; Assoc. Prof. Patbol., Harvard Med. Sch.* (4, 1956)

HIDALGO, JOHN, Ph.D. Cutter Labs., Berkeley 10, Calif. *Sec. Leader, Pharmacol. Research.* (3, 1958)

HIER, STANLEY W., Ph.D. Wilson Labs., 4221 S. Western Ave., Chicago 9, Ill. *Gen. Manager.* (2, 1950)

HIESTAND, WILLIAM A., Ph.D. Purdue U., Dept. of Biol., Lafayette, Ind. *Prof. of Physiol.* (1, 1947)

HIGGINBOTHAM, A. C., Ph.D. West Virginia U. Sch. of Med., Dept. of Microanatomy, Morgantown. *Assoc. Prof.* (1, 1955)

HIGGINS, GEORGE M., Ph.D. U. of Texas Dental Br., Houston 25. *Visiting Prof. of Histol.* (5R, 1948)

HIGGINS, HAROLD LEONARD, M.D. 322 Franklin St., Newton, Mass. (1, 1914; 5R, 1933)

HIGH, EDWARD G., Ph.D. Meharry Med. Coll., Nashville 8, Tenn. *Assoc. Prof. of Biochem.* (5, 1956)

HIGHMAN, BENJAMIN, M.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Med. Dir., PHS.* (4, 1947)

HIGHTOWER, NICHOLAS C., M.D., Ph.D. Scott and White Clinic, Temple, Tex. *Dir., Labs. for Clin. Invest. and Research.* (1, 1952)

HILDES, J. A., M.D. U. Manitoba Med. Coll., Dept. Physiol. ; Med. Res., Winnipeg 3, Canada. *Assoc. Prof. & Dir. Arctic Med. Res. Unit.* (1, 1957)

HILL, ARCHIBALD VIVIAN, C.H., O.B.E., Sc.D. 16 Bishopswood Rd., London N. 6, England. (1H, 1950)

HILL, CHARLES H., Ph.D. North Carolina State Coll., Dept. of Poultry Sci., Raleigh. *Assoc. Prof.* (5, 1958)

HILL, FREDRIC WILLIAM, Ph.D. Cornell U., Rice Hall, Ithaca, N. Y. *Prof. of Animal Nutrition and Poultry Husbandry.* (5, 1954)

HILL, HARRIS ERNEST, Ph.D. USPHS Hosp., Natl. Inst. of Mental Health, Addiction Res. Ctr., Lexington, Ky. *Sr. Res. Psychologist.* (3, 1958)

HILL, ROBERT L., Ph.D. U. of Utah Coll. of Med., Metabolic Lab., Salt Lake City 12. *Asst. Res. Prof.* (2, 1958)

HILL, ROBERT M., Ph.D. U. of Colorado Med. Ctr., Denver 20. *Prof. of Biochem.* (2, 1933)

HILL, WILLARD T., M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Assoc. Prof. of Patbol.* (4, 1956)

HILLEMAN, MAURICE, Ph.D. Merck Sharp & Dohme Research Labs., Merck & Co., West Point, Pa. *Dir.; Med. Bacteriologist (Virol.)* (6, 1949)

HILLIER, ATTIE YVONNE, Ph.D., M.D. Philadelphia Gen. Hosp., Philadelphia 4, Pa. (1, 1955)

HILTON, J. G., M.D. 115 E. 67th St., New York City 21. *Asst. Clin. Prof. Med., Columbia U.; Assoc. Physician, St. Luke's Hosp.* (1, 1957)

HILTON, JAMES G., Ph.D. U. of Mississippi Med. Ctr., Dept. of Pharmacol., Jackson. *Assoc. Prof.* (1, 1958; 3, 1955)

HIMWICH, HAROLD E., M.D. Galesburg State Research Hosp., Galesburg, Ill. *Research Dir.* (1, 1925; 3, 1957)

HIMWICH, WILLIAMINA A., Ph.D. Galesburg State Research Hosp., Galesburg, Ill. *Med. Research Assoc.* (1, 1947)

HINE, CHARLES H., Ph.D., M.D. U. of California Sch. of Med., Dept. of Pharmacol., San Francisco 22. *Assoc. Prof.* (3, 1947)

HINES, HARRY M., Ph.D. State U. of Iowa Coll. of Med., Dept. of Physiol., Iowa City. *Prof. and Head of Dept.* (1, 1928)

HINES, MARION, Ph.D. Emory U., Dept. of Anatomy, Atlanta 22, Ga. *Prof. of Exptl. Anatomy.* (1, 1932)

HINRICHS, M. A., Ph.D., M.D. 344 E. Quincy Rd., Riverside, Ill. *Visiting Prof. Health Educ., Roosevelt U., Chicago.* (1, 1928)

HINSEY, JOSEPH C., Ph.D., D.Sc. New York Hosp.-Cornell Med. Ctr., New York City 21. *Dir. and Prof. of Neuroanatomy.* (1, 1929)

HINSHAW, J. RAYMOND, M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Asst. Prof. in Surgery.* (4, 1956)

HIRS, C. H. W., Ph.D. Brookhaven Natl. Lab., Dept. of Biol., Upton, N.Y. *Assoc. Biochemist.* (2, 1955)

HIRSCH, FREDERIC G., M.D. 118 Dartmouth Dr., S.E., Albuquerque, N. Mex. *Biophysicist, Lovelace Fndn.* (1, 1951)

HIRSCHBERG, ERICH, Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Asst. Prof. of Biochem.* (2, 1958)

HIRSCHMANN, HANS, M.D., Ph.D. University Hosps., Cleveland 6, Ohio. *Assoc. Prof. of Biochem., Western Reserve U.* (2, 1946)

HIRST, GEORGE K., M.D. Public Health Research Inst., New York City 9. *Dir. and Chief, Div. of Infectious Diseases.* (6, 1951)

HISAW, FREDERICK L., Ph.D. Harvard U. Biol. Labs., Cambridge 38, Mass. *Prof. of Zool.* (1, 1932)

HITCHCOCK, DAVID I., Ph.D. Yale U., New Haven 11, Conn. *Assoc. Prof. of Physiol.* (2, 1930)

HITCHCOCK, FRED A., Ph.D. Ohio State U., Columbus. *Prof. of Physiol.* (1, 1927; 5, 1933)

HITCHCOCK, PHILIP, Ph.D., M.D. U. of Missouri Sch. of Med., Columbia. *Asst. Prof. of Anesthesiol.* (3, 1946)

HITCHINGS, GEORGE H., Ph.D. 50 Primrose Ave., Yonkers, N.Y. *Assoc. Research Dir., Burroughs-Wellcome Research Labs.* (2, 1942)

HJORT, AXEL M., M.D., Ph.D. 3341 S.W. 20th St., Fort Lauderdale, Fla. (2R, 1925)

HOAGLAND, HUDSON, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Exec. Dir.* (1, 1932)

HOAGLAND, MAHLON B., M.D. Massachusetts Gen. Hosp., Huntington Lab., Boston 14. *Assoc. in Med., Harvard Med. Sch.* (2, 1956)

HOBBY, GLADYS L., Ph.D. Chas. Pfizer & Co., Inc., 630 Flushing Ave., Brooklyn 6, N.Y. *Res. Microbiol., Exptl. Chemo-and Immuno-Therapy.* (6, 1946)

HOBERMAN, HENRY D., Ph.D., M.D. Albert Einstein Coll. of Med., New York City 61. *Prof. of Biochem.* (2, 1949)

HOCH, FREDERIC L., M.D. P. B. Brigham Hosp., Biophysics Res. Lab., Boston 15, Mass. *Assoc. in Med.; Assoc. in Med., Harvard Med. Sch.* (2, 1957)

HOCH, HANS, Ph.D. 1614 Greenleaf Lane, Charlottesville, Va. *Assoc. Prof. of Biophysics, Med. Coll. of Virginia.* (2, 1952)

HOCH-LIGETI, CORNELIA, M.D. U. of Virginia Sch. of Med., Dept. of Pathol., Charlottesville. *Asst. Prof.* (4, 1955)

HOCHSTER, ROLF M., Ph.D. Canada Dept. of Agric., Bacteriol. Div., Science Serv., Ottawa, Canada. *Head, Bacterial Metab.* (2, 1958)

HOCK, RAYMOND J., Ph.D. Arctic Aeromed. Lab., (Ladd AFB, Fairbanks, Alaska), APO 731, Seattle, Wash. *Res. Physiologist.* (1, 1954)

HOCKETT, ROBERT C., Ph.D. Tobacco Industry Research Comm., 150 E. 42nd St., New York City 17. *Assoc. Sci. Dir.* (2, 1950)

HODES, ROBERT, Ph.D. c/o Turner, 81 Chancery Lane, London, W.C. 1, England. (1, 1941)

HODGE, HAROLD CARPENTER, Ph.D. U. of Rochester Med. Ctr., Rochester 20, N.Y. *Prof. of Pharmacol. and Toxicol.* (2, 1937; 3, 1948)

HODGKIN, ALAN LLOYD, M.A. Physiological Lab., Cambridge, England. *Research Prof.* (1H, 1952)

HODGSON, EDWARD S., Ph.D. Columbia U., Dept. of Zool., New York City 27. *Assoc. Prof.* (1, 1956)

HOEFER, JACOB A., Ph.D. Michigan State U., East Lansing. *Prof. of Animal Husbandry.* (5, 1958)

HOEFER, PAUL F. A., Ph.D., M.D. Neurological Inst. of New York, 710 W. 168th St., New York City 32. *Assoc. Prof. of Neurol.* (1, 1945)

HOEKSTRA, JUSTIN B., Ph.D. Bristol Labs., Inc., Syracuse 1, N.Y. *Sr. Research Pharmacologist.* (3, 1956)

HOELSCHER, BERNHARD, M.D. Westendkrankenhaus, Berlin-Charlottenburg, Germany. (1, 1955)

HOERLEIN, ALVIN B., D.V.M., Ph.D. U. of Illinois Coll. of Veterinary Med., Urbana. *Prof. of Veterinary Pathol. & Hygiene.* (6, 1958)

HOFF, EBBE CURTIS, Ph.D., M.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Neurol. Sci.; Dean of Grad. Studies.* (1, 1933)

HOFF, HEBBEL E., Ph.D., M.D. Baylor U. Coll. of Med., Dept. of Physiol., Houston 25, Tex. *Hamblton Prof. & Chmn. of Dept.* (1, 1933)

HOFFBAUER, FREDERICK WILLIAM, M.D. U. of Minnesota Hosps., Dept. of Med., Minneapolis 14. *Assoc. Prof. of Med.* (4, 1952)

HOFFMAN, BRIAN F., M.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Prof. of Physiol.* (1, 1954)

HOFFMAN, WILLIAM SAMUEL, Ph.D., M.D. Sidney Hillman Health Ctr. of Chicago, 333 S. Ashland Blvd., Chicago 7, Ill. *Med. Dir.* (2, 1935)

HOFMANN, FREDERICK G., Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Asst. Prof.* (3, 1958)

HOFMANN, KLAUS, Ph.D. U. of Pittsburgh Med. Sch., Dept. of Biochem., Pittsburgh, Pa. *Prof. and Chmn. of Dept.* (2, 1947)

HOFSTEE, B. H. J., Ph.D. Palo Alto Med. Res. Fndn., 860 Bryant St., Palo Alto, Calif. *Sr. Biochemist.* (2, 1958)

HOGAN, ALBERT G., Ph.D. U. of Missouri, 105 Schweitzer Hall, Columbia. *Prof. Emer. of Animal Nutrition.* (2R, 1916; 5, 1933)

HOGBEN, C. ADRIAN M., M.D., Ph.D. George Washington U., Dept. of Physiol., Washington 5, D.C. *Prof.* (1, 1953; 3, 1957)

HOGG, JAMES F., Ph.D. U. of Michigan, Dept. of Biol. Chem., Ann Arbor. *Asst. Prof.* (2, 1958)

HOKIN, LOWELL E., M.D., Ph.D. U. of Wisconsin, Dept. of Physiol. Chem., Madison 6. *Asst. Prof.* (2, 1954)

HOLCK, HARALD G. O., Ph.D. U. of Nebraska, Ly-

man Hall 102, Lincoln 8. *Prof. Emer. of Pharmacol.; Consultant in Pharmacol.* (1R, 1935; 3, 1938)

HOLDEN, JOSEPH T., Ph.D. City of Hope Med. Ctr., Dept. of Biochem., Duarte, Calif. *Res. Assoc.* (2, 1958)

HOLLAENDER, ALEXANDER, Ph.D. Oak Ridge Natl. Lab., Oak Ridge, Tenn. *Dir., Biol. Div.* (1, 1947)

HOLLAND, WILLIAM CANNON, M.D. Vanderbilt U. Sch. of Med., Dept. of Pharmacol., Nashville 4, Tenn. *Assoc. Prof.* (3, 1951)

HOLLANDER, FRANKLIN, Ph.D. Mount Sinai Hosp., New York City 29. *Assoc. in Physiol.; Head, Gastroenterol. Res. Lab.* (1, 1942; 2, 1932)

HOLLANDER, VINCENT P., M.D., Ph.D. U. of Virginia Sch. of Med., Charlottesville. *Coordinator, U. Cancer Program.* (2, 1958)

HOLLEY, ROBERT W., Ph.D. U.S. Nutrition Lab., Cornell U., Dept. of Biochem., Ithaca, N. Y. *Chemist; Assoc. Prof.* (2, 1956)

HOLLINSHEAD, W. HENRY, Ph.D. Mayo Clinic, Rochester, Minn. *Head of Sec. of Anatomy; Prof. of Anatomy, Mayo Fndn.* (1, 1949)

HOLMAN, RALPH THEODORE, Ph.D. Hormel Inst., U. of Minnesota, Austin. *Prof. of Physiol. Chem.* (2, 1948; 5, 1957)

HOLMAN, RUSSELL LOWELL, M.D. Louisiana State U. Sch. of Med., New Orleans. *Prof. of Pathol.* (4, 1940)

HOLMES, JOSEPH H., M.D., D.Med.Sc. U. of Colorado Sch. of Med., Denver 20. *Prof. of Med.* (1, 1947)

HOLMES, JULIA O., Ph.D. 79 Old Town Rd., Amherst, Mass. (2, 1942; 5, 1936)

HOLT, JOSEPH PAYNTER, M.D., Ph.D. U. of Louisville, Heart Lab., 101 W. Chestnut St., Louisville 2, Ky. *Prof., Heart Research.* (1, 1942)

HOLT, L. EMMETT, JR., M.D. New York U. Coll. of Med., New York City 16. *Prof. of Pediatrics.* (2, 1930; 5, 1933)

HOLTKAMP, DORSEY E., Ph.D. Smith, Kline & French Labs., Philadelphia 1, Pa. *Endocrine-Metabolic Group Leader in Biochem.* (3, 1954)

HOMBURGER, FREDDY, M.D. Bio-Research Inst., Inc., 9 Commercial Ave., Cambridge 41, Mass. *Pres.* (4, 1957)

HOOBLER, ICIE MACY, Ph.D., Sc.D. 71 E. Ferry Ave., Detroit 2, Mich. *Consultant, Merrill-Palmer Sch.* (2, 1925; 5, 1933)

HOOBLER, SIBLEY W., M.D. U. of Michigan Hosp., Dept. of Med., Ann Arbor. *Assoc. Prof. of Int. Med.* (1, 1949)

HOOKER, DAVENPORT, Ph.D. 110 Livingston St., New Haven, Conn. *Prof. & Head, Anatomy (Med.), Emer., U. of Pittsburgh.* (1R, 1920)

HOOKER, SANFORD B., M.D. Bradford, Vt. (6R, 1918)

HOOVER, SAM R., Ph.D. 2017 Hillyer Pl., N. W., Washington 9, D. C. *Asst. Dir., Eastern Utiliza. Res. & Develop. Div., ARS, USDA.* (2, 1946)

HOPPE, JAMES O., Ph.D. Sterling-Winthrop Research Inst., Rensselaer, N. Y. *Research Pharmacologist.* (3, 1950)

HOPPERT, C. A., Ph.D. Michigan State U., Box 626, East Lansing. *Prof. of Biol. Chem.* (5, 1935)

HOPPS, HOPE E. B., M.S. Natl. Inst. of Allergy & Infectious Diseases, Rickettsial Biol. & Metab. Unit, NIH, Bethesda 14, Md. *Bacteriologist.* (6, 1958)

HOPPS, HOWARD C., M.D. U. of Texas Med. Br. Dept. of Pathol., Galveston. *Prof. and Chmn. of Dept.* (4, 1947; 6, 1946)

HORECKER, BERNARD L., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Lab. of Biochem. & Metab.* (2, 1947)

HORITA, AKIRA, Ph.D. U. of Washington Sch. of Med., Dept. of Pharmacol., Seattle 5. *Asst. Prof.* (3, 1958)

HORN, MILLARD J., Ph.D. 1811 Varnum St., N. E., Washington 18, D. C. *USDA, Chemist.* (2, 1949)

HOROWITZ, NORMAN H., Ph.D. California Inst. of Tech., Pasadena. *Prof. of Biol.* (2, 1946)

HORSFALL, FRANK L., JR., M.D., C.M. Rockefeller Inst. for Med. Res., New York City 21. *Vice Pres.; Physician-in Chief.* (6, 1937)

HORSTMANN, DOROTHY M., M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Assoc. Prof. of Prev. Med. & Pediatrics.* (6, 1951)

HORTON, RICHARD G., Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Chief, Toxicity Screening Br.* (1, 1948)

HORVATH, STEVEN M., Ph.D. Lankenau Hosp., Philadelphia 31, Pa. *Head, Dept. of Physiol.* (1, 1943)

HORWITT, BENJAMIN N., Ph.D. Mt. Sinai Hosp., Chicago 8, Ill. *Head, Div. of Biochem.* (2, 1953)

HORWITT, M. K., Ph.D. Elgin State Hosp., Elgin, Ill. *Dir., Research Lab.; Assoc. Prof., Biochem., U. of Ill. Coll. of Med.* (2, 1941; 5, 1949)

HORWITZ, ORVILLE, M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Asst. Prof. of Clin. Med.* (1, 1955)

HOSKINS, R. G., Ph.D., M.D. 86 Varick Rd., Waban 68, Mass. (1R, 1911)

HOTCHKISS, ROLLIN D., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1941)

HOTTLE, GEORGE A., Ph.D. Div. of Biologics Standards, Lab. of Viral Products, NIH, Bethesda 14, Md. *Chief, Testing Sec.* (6, 1958)

HOU, H. C., M.D. 372 Wu Kang Rd., Shanghai, China. *Prof. of Biochem. and Head of Dept.* (5, 1949)

HOUDE, R. W., M.D. 444 E. 68th St., New York City 21. *Asst. Prof. Med., Cornell U.; Assoc., Sloan-Kettering Inst., Mem. Cancer Ctr.* (3, 1953)

HOUSSAY, BERNARDO A., M.D. Viamonte 2790, Buenos Aires, Argentina. *Dir., Inst. Biol. and Exptl. Med.* (1H, 1942)

HOVE, EDWIN L., Ph.D. Div. of Research Grants, NIH, Bethesda 14, Md. *Biochemist.* (5, 1946)

HOWARD, EVELYN, Ph.D. Johns Hopkins Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Physiol.* (1, 1933)

HOWARD, JOHN EAGER, M.D. 1101 St. Paul St., Baltimore 2, Md. *Assoc. Prof. of Med., Johns Hopkins U.* (1, 1946)

HOWE, CALDERON, M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Microbiol.* (6, 1953)

HOWE, EUGENE E., Ph.D. R.D. 1, Bound Brook, N. J. *Dir. of Nutrition, Merck Inst. for Therap. Research.* (2, 1953)

HOWE, HOWARD A., M.D. Johns Hopkins U., 1901

East Madison St., Baltimore 5, Md. *Adjunct Prof. of Epidemiol.* (6, 1949)

HOWE, PAUL E., Ph.D. 3601 Connecticut Ave., N.W., Washington 8, D.C. *Nutrition Consultant.* (1R, 1913; 2R, 1909; 5R, 1933)

HOWELL, DAVID SANDERS, M.D. U. of Miami Sch. of Med., Miami, Fla. *Asst. Prof. of Med. and Dir. of Arthritis Training Prog.* (1, 1957)

HOWELL, STACEY F., Ph.D. Kirksville Coll. of Osteopathy and Surgery, Kirksville, Mo. *Prof. of Physiol. Chem.* (2, 1940)

HOWLAND, JOE W., Ph.D., M.D. U. of Rochester Sch. of Med., Rochester 20, N.Y. *Prof. Radiation Biol.* (1, 1949; 4, 1951)

HSIA, SUNG-LAN, Ph.D. St. Louis U. Sch. of Med., St. Louis 4, Mo. *Res. Asst. in Biochem.* (2, 1958)

HSIUNG, G. D., Ph.D. Yale U. Sch. of Med., Sec. of Epidemiol., New Haven 11, Conn. *Res. Assoc.* (6, 1958)

HUANG, H. T., Ph.D. Chas. Pfizer & Co., Inc., Brooklyn 6, N.Y. *Research Biochemist.* (2, 1953)

HUANG, KEE-CHANG, M.D., Ph.D. U. of Louisville Sch. of Med., Louisville 2, Ky. *Asst. Prof. of Pharmacol.* (1, 1957; 3, 1957)

HUBBARD, RUTH H., Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Research Fellow.* (2, 1953)

HUBBELL, REBECCA B., Ph.D. 107 Cottage St., Apt. 2A, New Haven 11, Conn. *Ed. Asst., J. Nutrition.* (2R, 1937; 5R, 1935)

HUDDLESTON, O. LEONARD, M.D., Ph.D. California Rehabilitation Ctr., 1 Pico Blvd., Santa Monica, Calif. *Med. Dir.* (1, 1936)

HUEBNER, ROBERT J., M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Chief, Lab. of Infectious Diseases.* (6, 1958)

HUENNEKENS, FRANK M., Ph.D. U. of Washington, Dept. of Biochem., Seattle 5. *Assoc. Prof.* (2, 1952)

HUEPER, WILHELM C., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief, Environ. Cancer Sec.* (4, 1940)

HUF, ERNST G., Ph.D., M.D. Med. Coll. of Virginia, Dept. of Physiol., Richmond 19. *Prof.* (1, 1949)

HUFF, JESSE W., Ph.D. Merck Sharp & Dohme, Div. of Merck & Co., Inc., West Point, Pa. *Dir., Research in Physiol. Chem.* (2, 1949)

HUFFMAN, C. F., Ph.D. Michigan State U., Anthony Hall, East Lansing. *Research Prof. and Prof. in Dairy Husbandry.* (5, 1937)

HUFFMAN, MAX N., Ph.D. Oklahoma Med. Research Fndn., Oklahoma City 4. *Prof. of Res. Biochem., U. of Oklahoma.* (2, 1947)

HUGGINS, CHARLES BRENTON, M.D. U. of Chicago, Chicago 37, Ill. *Prof. of Surgery.* (1, 1932)

HUGGINS, RUSSELL A., Ph.D. Baylor U. Coll. of Med., Houston 25, Tex. *Prof. of Pharmacol.* (1, 1952; 3, 1949)

HUGHES, JOHN RUSSELL, Ph.D. Meyer Mem. Hosp., Buffalo, N.Y. *Chief of Neurophysiol. & EEG; Asst. Prof. Neurophysiol., Buffalo Med. Sch.* (1, 1957)

HUGHES, JOSEPH, M.D. 111 N. 49th St., Philadelphia, Pa. *Assoc. Prof. Exptl. Neurol., U. of Pa.; Prof. Psychiatry, Woman's Med. Coll.* (1, 1936)

HUGHES, JOSIAH SIMPSON, Ph.D. Kansas State Coll., Manhattan. *Prof. of Chem.* (2, 1931; 5R, 1939)

HUGHES, THOMAS P., Ph.D. World Health Organization, Casilla 1167, Asuncion, Paraguay. *Public Health Lab. Consultant.* (6, 1934)

HUGHES, WALTER L., Ph.D. Brookhaven Natl. Lab., Med. Dept., Upton, N.Y. *Head, Div. of Biochem.* (2, 1955)

HULL, WAYLAND E., Ph.D. Duke U. Sch. of Med., Durham, N.C. *Asst. Prof. of Physiol.* (1, 1955)

HULPIEU, HAROLD R., Ph.D. Indiana U. Sch. of Med., Indianapolis 7. *Prof. of Pharmacol.* (3, 1939)

HUME, DAVID M., M.D. Med. Coll. of Virginia, Dept. of Surgery, Richmond. *Prof. and Chmn. of Dept.* (4, 1958)

HUMMEL, JOHN P., Ph.D. State U. of Iowa, Dept. of Biochem., Iowa City. *Assoc. Prof.* (2, 1952)

HUMMELER, KLAUS, M.D. Children's Hosp. of Philadelphia, Philadelphia 46, Pa. *Asst. Prof. in Virol.* (6, 1958)

HUMPHRIES, JAMES C., Ph.D. U. of Kentucky, Dept. of Bacteriol., Lexington. *Prof.* (6, 1950)

HUNDLEY, JAMES M., M.D. F.A.O., United Nations, New York City. (5, 1950)

HUNSCHER, HELEN A., Ph.D. Western Reserve U., Dept. of Home Econ., Cleveland 6, Ohio. *Head of Dept.* (5, 1934)

HUNT, CARLTON C., M.D. U. of Utah Coll. of Med., Salt Lake City. *Prof. of Physiol.* (1, 1950)

HUNTER, ANDREW, C.B.E., M.B. Hosp. for Sick Children, Toronto, Canada. *Research Assoc.; Prof. Emer. Pathol. Chem. U. of Toronto.* (2R, 1908)

HUNTER, F. R., Ph.D. U. of the Andes, Biol. Dept., Bogotá, Colombia. (1, 1952)

HUNTER, FRANCIS EDMUND, JR., Ph.D. Washington U. Med. Sch., St. Louis 10, Mo. *Assoc. Prof. of Pharmacol.* (2, 1946; 3, 1951).

HUNTER, JESSE E., Ph.D. Allied Mills, Inc., Research Div., Box 459, Libertyville, Ill. *Dir. of Research.* (5, 1936)

HUNTER, JOHN, Ph.D. Defense Res. Med. Labs., Box 62, Postal Sta. 'K', Toronto, Canada. *Chief, Applied Physiol. Sec.* (1, 1948)

HURLBERT, ROBERT B., Ph.D. U. of Texas, M. D. Anderson Hosp., Houston 25. *Asst. Biochemist.* (2, 1958)

HURWITZ, JERARD, Ph.D. New York U. Sch. of Med., Dept. of Microbiol., New York City 16. *Asst. Prof.* (2, 1957)

HURWITZ, LEON, Ph.D. Vanderbilt U. Sch. of Med., Dept. of Pharmacol., Nashville, Tenn. *Asst. Prof.* (3, 1956)

HUTCHENS, JOHN O., Ph.D. U. of Chicago, Dept. of Physiol., Chicago 37, Ill. *Prof. and Chmn. of Dept.* (1, 1947)

HUTCHEON, DUNCAN E., M.D., Ph.D. Seton Hall Coll. of Med., Jersey City 4, N.J. *Fellow in Med.* (3, 1954)

HUTCHINGS, BRIAN L., Ph.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Dir. of Biochem. Research.* (2, 1949)

HUTTRER, CHARLES P., Ph.D. Natl. Heart Inst., Grants and Training Br., NIH, Bethesda 14, Md. *Asst. Chief.* (1, 1957)

HWANG, KAO, M.D., Ph.D. Abbott Labs., North Chicago, Ill. *Group Leader in Pharmacol.* (3, 1953)

HYDE, JANE E., Ph.D. U. of California Med. Ctr., Dept. of Physiol., Los Angeles 24. *Asst. Research Physiologist.* (1, 1951)

HYMAN, CHESTER, Ph.D. U. of Southern California, Dept. of Physiol., Los Angeles 7. *Prof.* (1, 1948)

IAMPIETRO, P. F., Ph.D. QM Res. & Engin. Command, Natick, Mass. *Chief, Environ. Physiol. Sec.,* (1, 1958)

IKAWA, MIYOSHI, Ph.D. U. of California, Dept. of Biochem., Berkeley 4. (2, 1956)

IMAGAWA, DAVID T., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Asst. Prof. of Pediatrics and Infectious Diseases.* (4, 1957)

IMIG, CHARLES J., Ph.D. State U. of Iowa, Dept. of Physiol., Iowa City. *Assoc. Prof.* (1, 1955)

INGALLS, MABEL S., Ph.D. 860 Fifth Ave., New York City 21. *Assoc., Prof., Public Health Practice, Columbia U. Sch. Public Health.* (6, 1940)

INGRAHAM, LLOYD L., Ph.D. U. of California, Dept. of Agric. Biochem., Davis. *Asst. Prof.* (2, 1958)

INGRAHAM, RAYMOND CLIFFORD, Ph.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Physiol.* (1, 1938)

INGRAM, W. R., Ph.D. State U. of Iowa Coll. of Med., Dept. of Anatomy, Iowa City. *Prof. and Head of Dept.* (1, 1936)

INNES, JAMES R. M., Ph.D., D.Sc. Brookhaven Natl. Lab., Dept. of Biol., Upton, N. Y. *Pathologist.* (4, 1955)

IOB, VIVIAN, Ph.D. University Hosp., Kresge Med. Res. Bldg., R. 5556, Ann Arbor, Mich. *Instr., Dept. Surg., U. of Mich.* (2, 1957)

IPSEN, JOHANNES, JR., M.D. Massachusetts Dept. Public Health, Jamaica Plain. *Supt., Inst. of Labs.; Assoc. Prof. Public Health, Harvard.* (6, 1953)

IRISH, DON D., Ph.D. Dow Chemical Co., Midland, Mich. *Dir., Biochem. Research Lab.* (3, 1948)

IRVIN, ELINOR MOORE, Ph.D. U. of North Carolina Sch. of Med., Dept. of Biochem., Chapel Hill. *Guest Invest.* (2, 1951)

IRVIN, J. LOGAN, Ph.D. U. of North Carolina Sch. of Med., Dept. of Biochem., Chapel Hill. *Prof. and Chmn. of Dept.* (2, 1942)

IRVING, GEORGE WASHINGTON, JR., Ph.D. USDA, Agric. Research Serv., Washington 25, D.C. *Dept. Admin.* (2, 1946)

IRVING, LAURENCE, Ph.D. USPHS, P.O. Box 960, Anchorage, Alaska. *Biologist, Arctic Health Research Ctr.* (1, 1927)

IRWIN, M. R., Ph.D. U. of Wisconsin, Dept. of Genetics, Madison 6. *Prof.* (6, 1936)

IRWIN, SAMUEL, Ph.D. Schering Corp., Dept. of Pharmacol., Bloomfield, N. J. *Sr. Pharmacologist.* (3, 1955)

ISAACS, RAPHAEL, M.D. 104 S. Michigan Ave., Suite 630, Chicago 3, Ill. (4, 1928)

ISBELL, HARRIS, M.D. PHS Hosp., Lexington, Ky. *Dir., NIMH Addiction Research Ctr.* (3, 1949)

ISENBERGER, R. M., M.D. U. of Kansas Sch. of Med., Kansas City 12. *Prof. of Pharmacol.* (3, 1937)

ISHIZAKA, KIMISHIGE, M.D. Natl. Inst. of Health, Tokyo, Japan. *Chief, Div. of Immuno-serol.; Fulbright Res. Scholar, Calif. Inst. of Tech.* (6, 1958)

ITANO, HARVEY A., M.D., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Surgeon (PHS).* (2, 1953)

IVY, A. C., Ph.D., M.D. U. of Illinois, Chicago 12. *Distinguished Prof. Physiol. and Head, Dept. of Clin. Sci.* (1, 1919; 5, 1933)

IWAMOTO, HARRY K., Ph.D. Baxter Labs., Morton Grove, Ill. *Chief, Pharmacol.-Physiol. Sec.* (3, 1948)

IZQUIERDO, J. JOAQUIN, M.D. The University, Fac. of Med., Mexico City. *Prof. and Head, Dept. of Physiol.* (1, 1928)

JACKSON, DENNIS E., Ph.D., M.D. 144 Louis Ave., Cincinnati 19, Ohio. *Prof. Emer. Pharmacol., U. of Cincinnati Med. Coll.* (1R, 1910; 3R, 1912)

JACKSON, DUDLEY P., M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Med.* (1, 1955)

JACKSON, ELIZABETH B., A.B. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Virologist.* (6, 1949)

JACKSON, EUGENE L., Ph.D. 1407 Cummings Dr., Richmond 20, Va. *Vice Pres. & Dir. Sci. Develop., A. H. Robins Co., Inc.* (3, 1942)

JACKSON, RICHARD W., Ph.D. USDA, Northern Regional Res. & Develop. Div., Peoria, Ill. *Chief, Fermentation Lab.* (2, 1930; 5, 1933)

JACKSON, ROBERT L., M.D. U. of Missouri, Dept. of Pediatrics, Columbia. *Prof. and Chmn. of Dept.* (5, 1953)

JACOBS, LEON, Ph.D. Natl. Inst. of Allergy & Infectious Diseases, Lab. Trop. Diseases, NIH, Bethesda 14, Md. *Head, Sec. Protozoal Diseases.* (6, 1958)

JACOBS, MERKEL HENRY, Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Emer. Prof. of Gen. Physiol.* (1, 1919)

JACOBS, WALTER A., Ph.D. Rockefeller Inst., New York City 21. *Member Emer.* (2R, 1908; 3R, 1913)

JACOBSEN, ERIK, M.D. Medicinalco Ltd., 37, Prags Blvd., Copenhagen S., Denmark. *Research Dir.* (3, 1950)

JACOBSON, EDMUND, Ph.D., M.D. Lab. for Clin. Physiol., 55 E. Washington St., Chicago, Ill. *Dir.* (1, 1929)

JACOBSON, LEON O., M.D. U. of Chicago, Chicago 37, Ill. *Prof. of Med.; Dir. of Argonne Cancer Research Hosp., USAEC.* (4, 1953)

JAFFE, HENRY L., M.D. Hosp. for Joint Diseases, 1919 Madison Ave., New York City. *Dir. of Labs.* (4, 1925)

JAGER, BLAIR VALDEMAR, M.D. VA Hosp., Irving Ave. & University Pl., Syracuse, N. Y. (6, 1954)

JAHN, THEODORE LOUIS, Ph.D. U. of California, Los Angeles 24. *Chmn., Dept. of Zool.* (1, 1944)

JAILER, JOSEPH W., M.D., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Med.* (1, 1948)

JAKOBY, WILLIAM B., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Biochemist-Microbiologist.* (2, 1957)

JANDORF, BERNARD J., Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Chief, Biochem. Research Div.* (2, 1946)

JANES, RALPH G., Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Prof. of Anatomy.* (1, 1948)

JANOWITZ, HENRY D., M.D. Mt. Sinai Hosp., 1 E. 100th St., New York City 29. *Chief, Gastrointes-*

tinal Clinic. (1, 1951)

JANSEN, EUGENE F., B.A. USDA, Western Utilization Research Br., Albany 6, Calif. (2, 1947)

JAQUES, L. B., Ph.D. U. of Saskatchewan, Saskatoon, Canada. *Prof. and Head of Dept. of Physiol. and Pharmacol.* (1, 1943; 3, 1954)

JAQUES, WILLIAM E., M.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Prof. and Chmn. of Pathol.* (4, 1954)

JARCHO, LEONARD W., M.D. VA Hosp., 12th Ave. and E St., Salt Lake City 3, Utah. *Asst. Prof. of Med.; Chief, Med. Serv.* (1, 1951)

JARVIK, MURRAY E., M.D., Ph.D. Albert Einstein Coll. of Med., New York City 61. *Asst. Prof. of Pharmacol.* (3, 1957)

JASPER, HERBERT H., Ph.D., D.Sc., M.D. Montreal Neurol. Inst., 3801 University St., Montreal, Canada. *Prof. of Exptl. Neurol., McGill U.* (1, 1940)

JAWETZ, ERNEST, Ph.D., M.D. U. of California Med. Ctr., San Francisco 22. *Prof. of Microbiol.; Lect. in Med. and Pediatrics.* (6, 1950)

JEANLOZ, ROGER W., D.Sc. Massachusetts Gen. Hosp., Boston 14. *Assoc. Biochemist.* (2, 1954)

JENCKS, WILLIAM P., M.D. Brandeis U., Grad. Dept. of Biochem., Waltham, Mass. *Asst. Prof.* (2, 1958)

JENDEN, DONALD J., M.B., B.S. U. of California Med. Ctr., Dept. of Pharmacol., Los Angeles 24. *Assoc. Prof.* (3, 1955)

JENNESS, ROBERT, Ph.D. U. of Minnesota, Dept. of Agric. Biochem., St. Paul. *Prof.* (2, 1953)

JENNEY, ELIZABETH HOLDEN, M.S. Emory U., Div. of Basic Health Sci., Dept. of Pharmacol., Atlanta 22, Ga. *Instr.* (3, 1958)

JENNINGS, FRANK LAMONT, M.D. U. of Chicago, Dept. of Pathol., Chicago 37, Ill. *Asst. Prof.* (4, 1954)

JENNINGS, ROBERT B., M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Asst. Prof.* (4, 1956)

JENNINGS, ROBERT K., Ph.D. Rheumatic Fever Research Inst., 3026 S. California Ave., Chicago 8, Ill. *Member.* (6, 1957)

JENSEN, ELWOOD V., Ph.D. U. of Chicago, Dept. of Biochem. and Ben May Lab. for Cancer Research, Chicago 37, Ill. *Assoc. Prof.* (2, 1954)

JENSEN, HANS F., Ph.D. Army Med. Research Lab., Fort Knox, Ky. *Head, Biochem. Dept.* (2, 1929)

JENSEN, KEITH E., Ph.D. Chas. Pfizer & Co., Inc., Biologics Res. Dept., Terre Haute, Ind. (6, 1954)

JESAITIS, MARGERIS A., Sc.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (2, 1957)

JETER, WAYBURN S., Ph.D. State U. of Iowa, Dept. of Bacteriol., Iowa City. *Asst. Prof.* (6, 1955)

JOACHIMOGLU, GEORGE, M.D. 3A Lykeiou St., Athens, Greece. *Prof. of Pharmacol.* (3, 1951)

JOBLING, JAMES W., M.D. Suwanee Hotel, St. Petersburg, Fla. *Prof. Emer. of Pathol., Columbia U.* (4R, 1913)

JOCHIM, KENNETH E., Ph.D. U. of Kansas, Dept. of Physiol., Lawrence. *Prof. and Chmn. of Dept.* (1, 1942)

JOEL, WALTER, M.D. U. of Oklahoma Med. Ctr., Oklahoma City 4. *Prof. of Pathol.* (4, 1958)

JOHNSON, ARTHUR G., Ph.D. U. of Michigan Med. Sch., Dept. of Bacteriol., Ann Arbor. *Asst. Prof.* (6, 1957)

JOHNSON, B. CONNOR, Ph.D. U. of Illinois, Div. Animal Nutrition, 259 Animal Sci. Lab., Urbana. *Prof. Animal Biochem.* (2, 1947; 5, 1947)

JOHNSON, FRANK H., Ph.D. Princeton U., Dept. of Biol., Princeton, N. J. *Prof.* (1, 1942)

JOHNSON, J. RAYMOND, Ph.D. Creighton U. Sch. of Med., Dept. of Physiol. and Pharmacol., Omaha 2, Nebr. *Prof. and Dir.* (1, 1938)

JOHNSON, JOHN A., Ph.D. U. of Minnesota Med. Sch., Dept. of Physiol., Minneapolis 14. *Assoc. Prof.* (1, 1955)

JOHNSON, JOSEPH L., Ph.D., M.D. Howard U. Coll. of Med., Washington 1, D. C. *Dean.* (1, 1934)

JOHNSON, MARVIN J., Ph.D. U. of Wisconsin, Biochem. Bldg., Madison. *Prof. of Biochem.* (2, 1941)

JOHNSON, PAUL E., Ph.D. Natl. Acad. of Sciences-Natl. Res. Council, Washington 25, D. C. *Exec. Secy., Food Protection Committee.* (5, 1958)

JOHNSON, RALPH M., Ph.D. Detroit Inst. of Cancer Res., Detroit 1, Mich. *Res. Assoc.; Asst. Prof., Physiol. Chem., Wayne State U.* (2, 1952)

JOHNSON, ROBERT E., M.D., Ph.D. U. of Illinois Coll. of Lib. Arts & Sci., Dept. of Physiol., Urbana. *Head of Dept.* (1, 1944; 2, 1939; 5, 1946)

JOHNSON, S. R., Ph.D. M.F.A. Milling Co., Springfield, Mo. *Dir. of Nutrition.* (5, 1947)

JOHNSON, SHIRLEY A., Ph.D. Henry Ford Hosp., Detroit 2, Mich. *Assoc. in Labs.* (1, 1956)

JOHNSON, VICTOR, Ph.D., M.D. Mayo Fndn., Rochester, Minn. *Dir.* (1, 1933)

JOHNSON, WALTER H., Ph.D. Defence Research Med. Labs., Postal Sta. K, Toronto, Canada. *Defence Sci. Officer.* (1, 1955)

JOHNSTON, CHARLES G., M.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Chmn., Div. of Surgery.* (1, 1933)

JOHNSTON, ELBERT FELTON, Ph.D. USDA, Agric. Research Ctr., Beltsville, Md. *Physiologist. (Animal).* (1, 1957)

JOHNSTON, FRANCES A., Ph.D. Cornell U., Ithaca, N. Y. *Prof. of Food and Nutrition.* (5, 1948)

JOHNSTON, MARGARET W., Ph.D. 1761 Westridge Rd., Ann Arbor, Mich. (2, 1930)

JOHNSTON, ROBERT B., Ph.D. U. of Nebraska, Lincoln 8. *Assoc. Prof. of Chem.* (2, 1957)

JOLLIFFE, NORMAN, M.D. 910 Park Ave., New York City 21. *Dir., Bur. of Nutrition.* (1, 1932; 5, 1957)

JONAS, HERBERT, Ph.D. U. of Minnesota Coll. of Pharmacy, Minneapolis 14. (1, 1956)

JONES, E. ELIZABETH, Ph.D. Wellesley Coll., Dept. of Zool. and Physiol., Wellesley 81, Mass. *Prof. of Zool.* (4, 1956)

JONES, HARDIN B., Ph.D. U. of California, Berkeley 4. *Prof., Med. Physics & Physiol.; Asst. Dir., Donner Lab.* (1, 1949)

JONES, JAMES H., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Biochem.* (2, 1928; 5, 1933)

JONES, KENNETH K., Ph.D. Clin. Research Labs., 433 N. Tucson Blvd., Tucson, Ariz. *Dir.* (1R, 1936)

JONES, L. MEYER, D.V.M., Ph.D. Iowa State Coll., Ames. *Prof. of Veterinary Physiol. and Pharmacol.* (3, 1953)

JONES, MARY ELLEN, Ph.D. Brandeis U., Dept. of Biochem., Waltham, Mass. *Asst. Prof.* (2, 1955)

JONES, RALPH, JR., M.D. U. of Miami Sch. of Med., Dept. of Med., Miami 36, Fla. *Prof. and Chmn. of Dept.* (3, 1950)

JONES, RUSSELL S. U. of Utah Coll. of Med., Dept. of Pathol., Salt Lake City. (4, 1952)

JORDAN, WILLIAM S., M.D. U. of Virginia Sch. of Med., Charlottesville. *Prof. of Preventive Med.; Prof. of Med.* (6, 1955)

JOSEPH, NORMAN R., Ph.D. U. of Illinois, Chicago 12, Ill. *Assoc. Prof. of Chem.* (2, 1947)

JOSLIN, ELLIOTT P., M.D., 170 Pilgrim Rd., Boston 15, Mass. *Pres., Diabetes Fndn.* (5R, 1933)

JOSLYN, MAYNARD A., Ph.D. U. of California, Berkeley 4. *Prof. of Food Tech.; Biochemist, Calif. Agric. Expt. Sta.* (2, 1956)

JUKES, THOMAS HUGHES, Ph.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Head, Nutrition and Physiol. Sec.* (2, 1935; 5, 1938)

JUNG, FREDERIC THEODORE, Ph.D., M.D. American Med. Assn., Chicago, Ill. *Asst. Editor, J.A.M.A.* (1, 1930)

JUNGCK, EDWIN C., Ph.D., M.D. Med. Coll. of Georgia, Dept. of Endocrinol., Augusta. (1, 1952)

JUNGEBLUT, CLAUS W., M.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Bacteriol.* (4, 1929; 6, 1926)

JUNI, ELLIOT, Ph.D. Emory U., Dept. of Bacteriol., Atlanta 22, Ga. *Assoc. Prof.* (2, 1954)

KABAT, ELVIN A., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Microbiol.* (2, 1940; 6, 1943)

KABAT, HERMAN, Ph.D., M.D. State of Rhode Island, Dept. of Social Welfare, Providence 3, R.I. (1, 1941)

KAESBERG, PAUL, Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Assoc. Prof.* (2, 1958)

KAGAN, B. M., M.D. Cedars of Lebanon Hosp., Los Angeles 29, Calif. *Dir., Dept. of Pediatrics; Clin. Prof. of Pediatrics, UCLA.* (5, 1953)

KAGAN, IRVING G., Ph.D. Communicable Disease Ctr., Atlanta, Ga. *Chief, Helminthology Unit, Microbiol. Sec.* (6, 1955)

KAGAWA, CHARLES M., Ph.D. G. D. Searle & Co., Div. of Biological Res., Chicago 80, Ill. *Res. Biologist.* (3, 1958)

KAHN, JULIUS B., JR., Ph.D. U. of Cincinnati Coll. of Med., Dept. of Pharmacol., Cincinnati 19, Ohio. *Asst. Prof.* (3, 1954)

KAHN, REUBEN L., Sc.D. U. of Michigan Hosp., Kresge Med. Res. Bldg., Ann Arbor. *Prof. of Serol. of Syphilis; Chief of Serol. Lab.* (4, 1934; 6, 1919)

KALCKAR, HERMAN M., M.D., Ph.D. Johns Hopkins U., Sch. of Biol., Baltimore, Md. *Prof.* (2, 1942)

KALLIO, REINO E., Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Assoc. Prof. of Bacteriol.* (2, 1957)

KALMAN, SUMNER M., M.D. Stanford U. Sch. of Med., Dept. of Pharmacol., San Francisco 15, Calif. *Asst. Prof.* (3, 1956)

KALNITSKY, GEORGE, Ph.D. State U. of Iowa, Dept. of Biochem., Iowa City. *Prof.* (2, 1948)

KALOW, WERNER, M.D. U. of Toronto, Dept. of Pharmacol., Toronto 5, Canada. *Assoc. Prof.* (3, 1951)

KALTER, S. S., Ph.D. Communicable Disease Ctr., P.O. Box 185, Chamblee, Ga. *Chief, Virus Diagnostic Methodol. Unit.* (6, 1950)

KAMEN, MARTIN D., Ph.D. Brandeis U., Waltham, Mass. *Prof. of Biochem.* (2, 1946)

KAMIJO, KAZUYA, M.D., Ph.D. Showa Med. Coll., Shinagawa-ku, Tokyo, Japan. *Prof. in Pharmacol.* (3, 1953)

KAMIN, HENRY, Ph.D. Duke Hosp., Durham, N.C. *Asst. Prof. Duke U. Sch. of Med.; Radio-Isotope Unit, VA Hosp.* (2, 1953)

KAMM, OLIVER, Ph.D. 360 Carver St., Grosse Pointe 36, Mich. *Consultant in field of Medicinal Prod.* (2R, 1928)

KANDEL, ALEXANDER, Ph.D. Smith, Kline & French Labs., Research & Develop. Div., Philadelphia 1, Pa. (3, 1956)

KANTER, GERALD S., Ph.D. Albany Med. Coll., Dept. of Physiol., Albany 8, N.Y. *Assoc. Prof.* (1, 1955)

KAO, CHIEN-YUAN, M.D. State U. of New York Coll. of Med., Brooklyn 3. *Asst. Prof. of Pharmacol.* (1, 1957)

KAO, FREDERICK F., Ph.D., M.D. State U. of New York Coll. of Med., Dept. of Physiol., Brooklyn 3. *Asst. Prof.* (1, 1956)

KAPLAN, ALBERT S., Ph.D. Albert Einstein Med. Ctr., Div. of Microbiol., Philadelphia 41, Pa. (6, 1958)

KAPLAN, ALEX, Ph.D. Children's Hosp., San Francisco, Calif. (1, 1951)

KAPLAN, HAROLD M., Ph.D. 510 W. Main St., Carbondale, Ill. *Prof. of Physiol., Southern Illinois U.* (1, 1954)

KAPLAN, HENRY S., M.D. Stanford U. Sch. of Med., Dept. of Radiol., San Francisco 15, Calif. *Prof.* (4, 1956)

KAPLAN, J. GORDIN, Ph.D. Dalhousie U., Dept. of Physiol., Halifax, Canada. *Assoc. Prof.* (1, 1956)

KAPLAN, MELVIN H., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Asst. Prof. of Med.* (6, 1956)

KAPLAN, NATHAN O., Ph.D. Brandeis U., Waltham, Mass. *Prof. of Biochem.* (2, 1949)

KARCZMAR, ALEXANDER G., Ph.D. Loyola U., Stritch Sch. of Med., Dept. of Pharmacol. & Therap., Chicago 12, Ill. *Prof. and Chmn.* (3, 1953)

KAREL, LEONARD, Ph.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Chief, Extramural Programs.* (3, 1947)

KARNOFSKY, DAVID A., M.D. Sloan-Kettering Inst., New York City 21. *Member.* (3, 1956)

KARNOVSKY, MANFRED L., Ph.D. Harvard Med. Sch., Biophysical Lab., Boston 15, Mass. *Asst. Prof., Biol. Chem.* (2, 1951)

KARP, DOROTHY, Ph.D. Baylor U. Coll. of Med., Texas Med. Ctr., Houston. *Instr. in Pharmacol.* (3, 1956)

KARPOVICH, PETER V., M.D. Springfield Coll., Springfield, Mass. *Prof. of Physiol.* (1, 1942)

KARSHAN, MAXWELL, Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Assoc. Prof.* (2, 1939)

KARSNER, HOWARD T., M.D. Dept. of the Navy, Bur. of Med. and Surgery, Washington 25, D.C.

Med. Research Advisor. (4R, 1913)

KARUSH, FRED, Ph.D. U. of Pennsylvania Sch. of Med., Dept. of Pediatrics, Philadelphia 46. *Prof. of Immunochem.* (2, 1957; 6, 1952)

KARZON, DAVID T., M.D. Children's Hosp., 219 Bryant St., Buffalo 22, N.Y. *Asst. Prof. of Pediatrics and of Virol.* (6, 1953)

KASS, E. H., M.D., Ph.D. Boston City Hosp., Boston 18, Mass. *Assoc. Dir., Mallory Inst.; Assoc. Prof. Bact. & Immunol., Harvard.* (6, 1955)

KASSELL, BEATRICE, Ph.D. Marquette U. Sch. of Med., Milwaukee 3, Wis. *Assoc. in Biochem.* (2, 1957)

KASTELIC, JOSEPH, Ph.D. U. of Illinois, Div. of Animal Nutrition, Urbana. *Prof.* (5, 1957)

KATSH, SEYMOUR, Ph.D. U. of Colorado Med. Ctr., Denver 20. *Asst. Prof. of Pharmacol.* (1, 1956)

KATZ, JOSEPH, Ph.D. Cedars of Lebanon Hosp., Inst. of Med. Research, Los Angeles, Calif. *Research Assoc.* (2, 1955)

KATZ, LOUIS NELSON, M.D. Michael Reese Hosp., 2900 Ellis Ave., Chicago, Ill. *Dir., Cardiovasc. Dept., Med. Res. Inst.* (1, 1924)

KATZMAN, PHILIP A., Ph.D. St. Louis U. Sch. of Med., St. Louis 4, Mo. *Prof. of Biochem.* (2, 1935)

KAUFMAN, NATHAN, M.D. City Hosp., 3395 Scranton Rd., Cleveland 9, Ohio. *Pathologist-in-Charge; Assoc. Prof. Pathol., Western Reserve U.* (4, 1953)

KAUFMAN, SEYMOUR, Ph.D. Natl. Inst. for Mental Health, NIH, Bethesda 14, Md. (2, 1953)

KAULBERSZ, JERZY, Ph.D., M.D. Collegium Medicum, Grzegorzecka 16, Cracow, Poland. *Prof. of Physiol.* (1, 1944)

KAUNITZ, HANS, M.D. Columbia U., Coll. of P. & S. New York City 32. *Research Assoc. in Pathol.* (4, 1947)

KAY, ERNEST R. M., Ph.D. Dalhousie U. Med. Sch., Dept. of Biochem., Halifax, Canada. *Asst. Prof.* (2, 1957)

KAZAL, L. A., Ph.D. Jefferson Med. Coll., Cardeza Fndn., Philadelphia 7, Pa. *Head, Sec. on Blood & Plasma Fractionation; Asst. Prof. Physiol.* (2, 1947)

KEARNEY, EDNA B., Ph.D. Edsel B. Ford Inst. for Med. Res., Dept. of Biochem., Henry Ford Hosp., Detroit 2, Mich. (2, 1953)

KEASLING, HUGH H., Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Assoc. Prof. of Pharmacol.* (3, 1953)

KEATS, ARTHUR S., M.D. Jefferson Davis Hosp., Houston 3, Tex. *Prof. of Anesthesiol., Baylor U. Coll. of Med.* (3, 1951)

KEHOE, ROBERT A., M.D. U. of Cincinnati Coll. of Med., Cincinnati 19, Ohio. *Dir., Dept. Prev. Med. & Indust. Health.* (1, 1940)

KEIGHLEY, GEOFFREY, Ph.D. California Inst. of Tech., Pasadena. *Sr. Research Fellow.* (1, 1954)

KEILES, ELSA ORENT, D.Sc. Div. of Research Grants, NIH, Bethesda 14, Md. *Biochemist.* (2, 1935; 5, 1935)

KEITH, NORMAN M., M.D. Mayo Clinic, Rochester, Minn. *Consulting Phys. Emer., Med.; Prof. Med. Emer., Mayo Fndn.* (1, 1930; 3R, 1932; 4, 1924)

KEITH, T. B., Ph.D. U. of Idaho, Dept. of Animal Husbandry, Moscow. *Prof.* (5, 1941)

KELLAWAY, PETER, Ph.D. Baylor U. Coll. of Med., Houston, Tex. *Assoc. Prof. Neurol. and Physiol.; Dir., Lab. Clin. Electrophysiol.* (1, 1947)

KELLER, ALLEN D., Ph.D. Army Med. Research Lab., Fort Knox, Ky. (1, 1931)

KELLER, ELIZABETH B., Ph.D. Massachusetts Gen. Hosp., Huntington Lab., Boston 14. *Res. Assoc. in Med., Harvard Med. Sch.* (2, 1955)

KELLNER, AARON, M.D. Cornell U. Med. Coll., Dept. of Pathol., New York City 21. *Assoc. Prof.; Dir. Central Labs., N. Y. Hosp.* (4, 1949)

KELLOGG, RALPH H., M.D., Ph.D. U. of California Med. Ctr., San Francisco 22. *Asst. Prof. of Physiol.* (1, 1956)

KELLY, LOLA S., Ph.D. U. of California, Donner Lab., Berkeley. 4. *Research Assoc. in Med. Physics.* (1, 1956)

KELSEY, F. ELLIS, Ph.D. U. of South Dakota, Vermillion. *Prof. of Pharmacol.; Chmn., Depts. of Physiol. & of Pharmacol.* (3, 1941)

KELSEY, FRANCES KATHLEEN O., Ph.D., M.D. 19 S. University Ave., Vermillion, S. Dak. (3, 1941)

KEMMERER, A. R., Ph.D. U. of Arizona, Dept. of Agric. Biochem., Tucson. *Head of Dept.* (5, 1946)

KEMPE, C. HENRY, M.D. U. of Colorado Med. Ctr., Dept. of Pediatrics, Denver 20. *Prof. and Head of Dept.* (6, 1951)

KEMPNER, WALTER, M.D. Duke U. Sch. of Med., Durham, N.C. *Prof. of Med.* (1, 1940)

KENDALL, EDWARD C., Ph.D. 3 Queenston Pl., Princeton, N. J. *Visiting Prof. in Chem., Forrestal Res. Ctr.,* (1, 1916; 2R, 1913; 4R, prior to 1920)

KENDALL, FORREST E., Ph.D. 240-06 53rd Ave., Douglaston, N. Y. *Prof. of Biochem., Columbia U.* (6, 1943)

KENNARD, MARGARET A., M.D. Mental Health Research Inst., Fort Steilacoom, Wash. (1, 1934)

KENNEDY, CORNELIA, Ph.D. 2700 W. Robbins St., Minneapolis 10, Minn. *Assoc. Prof. Emer., Agric. Biochem., U. of Minn.* (2R, 1924; 5R, 1934)

KENNEDY, EUGENE P., Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Prof.* (2, 1953)

KENNEDY, THOMAS J., JR., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Research Assoc., Lab. of Kidney and Electrolyte Metab.* (1, 1953)

KENSLER, CHARLES J., Ph.D. Boston U. Sch. of Med., Dept. of Pharmacol. and Exptl. Therap., Boston, Mass. *Prof. and Chmn.* (3, 1949)

KENT, JOHN F., Ph.D. New York State Health Dept., Div. of Labs. and Research, Albany 1. *Assoc. Res. Scientist.* (6, 1947)

KENT, SIDNEY PAGE, M.D. U. of Alabama Med. Ctr., Dept. of Pathol., Birmingham 3. *Assoc. Prof.* (4, 1957)

KENTON, HAROLD B., Ph.D. New England Deaconess Hosp., Boston, Mass. *Bacteriologist and Dir. of Blood Bank.* (6, 1934)

KENYON, ALLAN T., M.D. U. of Chicago, Div. of Biol. Sci., Chicago 37, Ill. *Prof. of Med.* (3, 1940)

KERESZTESY, JOHN C., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Sec. on Fractionation and Isolation.* (2, 1941; 5, 1945)

KERR, STANLEY E., Ph.D. American U. of Beirut, Beirut, Lebanon. *Prof. of Biol. Chem.* (2, 1937)

KERR, W. J., M.D. Kerrydale Ranch, Box 338, Blue

Lake, Humboldt County, Calif. (3R, 1930)

KERTESZ, ZOLTAN I., Ph.D. New York State Agric. Expt. Sta., Cornell U., Geneva. *Prof. of Chem.* (2, 1958)

KESSLER, RICHARD HOWARD, M.D. Cornell U. Med. Coll., Dept. of Physiol., New York City 21. *Instr.* (1, 1958)

KESTEN, HOMER D., M.D. Columbia U. Coll. of P. & S., New York City 32. *Assoc. Prof. of Patbol.; Dir. of Labs., White Plains Hosp.* (4, 1931)

KESTON, ALBERT S., Ph.D. New York U. Coll. of Med., New York City 16. *Asst. Prof., Dept. of Biochem.* (2, 1950)

KETY, SEYMOUR S., M.D. Natl. Inst. of Mental Health, NIH, Bethesda 14, Md. *Chief, Lab. of Clin. Sci.* (1, 1949; 3, 1945)

KEYL, ALEXANDER C., Ph.D. Northwestern U. Sch. of Med., Chicago 11, Ill. *Asst. Prof. of Pharmacol.* (3, 1957)

KEYS, ANCEL, Ph.D. U. of Minnesota, Stadium Gate 27, Minneapolis 14. *Prof. and Dir. of Lab. of Physiol. Hygiene.* (1, 1939; 2, 1936)

KHORANA, H. GOBIND, Ph.D. British Columbia Res. Council, U. of British Columbia, Vancouver, Canada. *Head, Organic Chem. Sec.* (2, 1957)

KIBRICK, ANDRE C., Ph.D. VA Hosp., 408 First Ave., New York City 10. *Biochemist.* (2, 1950)

KIDD, JOHN G., M.D. Cornell U. Med Coll., New York City 21. *Prof. of Pathol.* (4, 1937; 6, 1937)

KIDDER, GEORGE W., Ph.D. Amherst Coll., Amherst, Mass. *Stone Prof. of Biol.* (2, 1949)

KIELLEY, RUTH K., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1957)

KIELLEY, W. WAYNE, Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1953)

KIES, MARIAN W., Ph.D. Natl. Inst. of Mental Health, Lab. of Clin. Sci., NIH, Bethesda 14, Md. *Chief, Sec. on Biochem.* (2, 1948)

KIK, M. C., Ph.D. U. of Arkansas Coll. of Agric., Fayetteville. *Prof. of Agric. Chem.* (5, 1942)

KILBORN, L. G., M.D., Ph.D. U. of Hong Kong, Dept. of Physiol., Hong Kong. *Prof.* (1, 1928)

KILBOURNE, EDWIN DENNIS, M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof., Public Health & Prev. Med.* (6, 1953)

KILHAM, LAWRENCE, M.D. Natl. Insts. of Health, Bethesda 14, Md. *Med. Dir., PHS.* (6, 1950)

KILLAM, EVA KING, Ph.D. U. of California Coll. of Med., Dept. of Anatomy, Los Angeles 24. *Research Pharmacologist.* (3, 1953)

KILLAM, KEITH F., JR., Ph.D. U. of California Med. Ctr., Dept. of Pharmacol., Los Angeles 24. *Assoc. Res. Pharmacologist.* (3, 1958)

KIMELDORF, DONALD J., Ph.D. U.S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Head, Physiol.-Phychol. Br.* (1, 1955)

KIMMEL, JOE R., M.D., Ph.D. U. of Utah, Metabolic Lab., Salt Lake City. *Asst. Research Prof. of Biochem.* (2, 1957)

KIMURA, EUGENE T., Ph.D. Abbott Labs., Dept. of Pharmacol., North Chicago, Ill. *Pharmacologist.* (3, 1954)

KIMURA, KAZUO K., Ph.D., M.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Chief, Exptl. Med. Br.* (3, 1950)

KINARD, F. W., Ph.D., M.D. Med. Coll. of South Carolina, Charleston 16. *Prof. of Physiol.* (1, 1947)

KING, BARRY G., Ph.D. Operations Research, Inc., 8605 Cameron St., Silver Spring, Md. *Research Exec.* (1, 1938)

KING, CHARLES GLEN, Ph.D. Nutrition Fndn., Inc., 99 Park Ave., New York City. *Exec. Dir.; Prof. Chem., Columbia U.* (2, 1931; 5, 1933)

KING, HENRY EUGENE, Ph.D. Tulane U. Sch. of Med., Dept. of Psychiatry and Neurol., New Orleans 12, La. *Assoc. Prof.* (1, 1950)

KING, JOSEPH T., M.D., Ph.D. U. of Minnesota Med. Sch., 314 Millard Hall, Minneapolis. *Prof. of Physiol.* (1, 1931)

KING, THEODORE O., Ph.D. Johnson & Johnson Res. Fndn., New Brunswick, N. J. *Sr. Pharmacologist.* (3, 1958)

KING, TSOO E., Ph.D. Molteno Inst., U. of Cambridge, Cambridge, England. (2, 1952)

KINNEY, THOMAS D., M.D. Cuyahoga County Hosp., Cleveland 9, Ohio. *Dir. of Patbol.; Prof. of Pathol., Western Reserve U.* (4, 1950)

KINOSHITA, JIN H., Ph.D. Howe Lab., 243 Charles St., Boston 14, Mass. *Asst. Prof. of Biol. Chem., Harvard Med. Sch.* (2, 1958)

KINOSITA, RIOJUN, Ph.D., M.D. City of Hope Med. Ctr., Duarte, Calif. *Chmn., Dept. of Cytol. and Genetics.* (4, 1955)

KINSELL, LAURANCE W., M.D. Highland-Alameda County Hosp., Inst. for Metabolic Res., Oakland 6, Calif. *Dir.* (5, 1958)

KINTER, WILLIAM BOARDMAN, Ph.D. Harvard Med. Sch., Dept. of Physiol., Boston 15, Mass. *Assoc.* (1, 1957)

KINSEY, V. EVERETT, Ph.D. 690 Mullett St., Detroit 26, Mich. *Asst. Dir. of Res., Kresge Eye Inst.* (2, 1957)

KIRCH, ERNST R., Ph.D. U. of Illinois, 833 S. Wood St., Chicago 12. *Prof. of Chem.* (2, 1948)

KIRCHHOF, ANTON C., M.D 758 S. View Rd., Oswego, Oreg. *Anesthesiologist, Providence Hosp., Portland.* (3, 1947)

KIRK, JOHN E., M.D. Washington U. Sch. of Med., St. Louis 9, Mo. *Research Dir., Gerontol.* (2, 1950)

KIRK, PAUL L., Ph.D. U. of California, Berkeley 4. *Prof. of Criminalistics.* (2, 1933)

KIRKWOOD, SAM, Ph.D. U. of Minnesota, Dept. of Agric. Biochem., Inst. of Agric., St. Paul 1 (2, 1954)

KIRSCHBAUM, ARTHUR, M.D., Ph.D. Baylor U., Coll. of Med., Dept. of Anatomy, Houston, Tex. *Prof. and Chmn. of Dept.* (4, 1948)

KISCH, BRUNO, M.D. Yeshiva U., New York City 61. *Prof. of Biochem.* (1, 1943)

KIT, SAUL, Ph.D., M.D. Anderson Hosp. and Tumor Inst., Texas Med. Ctr., Houston. *Assoc. Biochemist.* (2, 1957)

KITCHEN, STUART F., M.D., M.P.H. Dept. of Natl. Health and Welfare, Lab. of Hygiene, Ottawa, Canada. (6, 1953)

KITZINGER, CHARLOTTE, M.D. Naval Med. Res. Inst., Bethesda 14, Md. *Med. Officer.* (1, 1958)

KLEBANOFF, SEYMOUR J., M.D., Ph.D. Rockefeller Inst. for Med. Res., New York City 21. *Guest Invest.* (2, 1958)

KLEEMAN, CHARLES R., M.D. VA Hosp., Los Angeles 25, Calif. *Chief, Metabolic Serv.; Assoc. Clin. Prof. Med., U. of Calif.* (1, 1957)

KLEIBER, M., D.Sc. U. of California, Davis. *Prof. of Animal Husbandry.* (1, 1943; 5, 1933)

KLEIN, J. RAYMOND, Ph.D. Brookhaven Natl. Lab., Upton, N. Y. *Sr. Biochemist, Dept. of Biol.* (2, 1941)

KLEIN, MICHAEL, Ph.D. U. of Tennessee Med. Units, Dept. of Anatomy, Memphis 3. *Asst. Prof.* (4, 1954)

KLEINER, ISRAEL S., Ph.D. New York Med. Coll., New York City 29. *Prof. and Dir., Dept. of Biochem.* (1, 1911; 2R, 1912; 3R, 1912; 5R, 1933)

KLEINERMAN, JEROME, M.D. St. Luke's Hosp., Cleveland 4, Ohio. *Assoc. Dir., Dept. Med. Res.; Asst. Prof. Pathol., Western Reserve U.* (4, 1954)

KLEITMAN, NATHANIEL, Ph.D. U. of Chicago, Chicago 37, Ill. *Prof. of Physiol.* (1, 1923)

KLEMPERER, FRIEDRICH W., M.D. VA Hosp., Sunmount, N. Y. *Chief of Med. Serv.* (2, 1941)

KLETZIEN, SEYMOUR W., Ph.D. 319 S. Chester Rd., Swarthmore, Pa. *Biochemist, Cooper Hosp., Camden, N. J.* (5, 1933)

KLINE, DANIEL L., Ph.D. Yale Med. Sch., Dept. of Physiol., New Haven, Conn. *Assoc. Prof.* (1, 1955)

KLINE, O. L., Ph.D. Food and Drug Admin., Dept. of HEW, Washington 25, D. C. *Biochemist.* (5, 1936)

KLINE, RAYMOND F., Ph.D. 6832 Knollwood Dr., Dayton 32, Ohio. *Res. Physiologist, Aero Med. Lab., Wright-Patterson AFB.* (1, 1946)

KLITGAARD, HOWARD M., Ph.D. Marquette U. Sch. of Med., Dept. of Physiol., Milwaukee 3, Wis. *Asst. Prof.* (1, 1956)

KOLTZ, IRVING M., Ph.D. Northwestern U., Evanston, Ill. *Prof. of Chem.* (2, 1947)

KLUVER, HEINRICH, Ph.D. U. of Chicago, Chicago, 37, Ill. *Sewell L. Avery Distinguished Serv. Prof. of Biol. Psychol.* (1, 1935)

KNIGHT, C. ARTHUR, Ph.D. U. of California, Biochem. and Virus Lab., Berkeley 4. *Prof. of Biochem.; Prof. of Virol.* (2, 1946)

KNISELY, MELVIN H., Ph.D. Med. Coll. of South Carolina, Dept. of Anatomy, Charleston. *Chmn.* (1, 1949)

KNOBIL, ERNEST, Ph.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Physiol.* (1, 1955)

KNODT, CLOY B., Ph.D. Cargill, Inc., Cargill-Nutrena Res. Farm, Elk River, Minn. *Dir.* (5, 1954)

KNOEFEL, PETER K., M.D. U. of Louisville, Dept. of Pharmacol., Louisville 2, Ky. *Prof. and Chmn. of Dept.* (3, 1934)

KNOWLTON, FRANK P., M.D. Syracuse U. Coll. of Med., Syracuse, N. Y. *Prof. Emer. of Physiol.* (1R, 1911)

KNOWLTON, G. CLINTON, Ph.D. Greenville, Ga. (1, 1938)

KNOX, W. EUGENE, M.D. New England Deaconess Hosp., Boston 15, Mass. *Asst. Prof. of Biol. Chem., Harvard Med. Sch.* (2, 1948)

KNUDSON, ARTHUR, Ph.D., D.Sc. U. of Kentucky-U. of Indonesia, Post Box 54, Bogor, Indonesia. *Prof. of Biochem.* (2R, 1919; 5, 1936)

KOBAYASHI, YOSHITO, M.D. U. of Tokyo, Faculty of Med., Bunkyo-ku, Tokyo, Japan. *Prof. of Pharmacol.* (3, 1953)

KOBER, PHILIP A., B.S. Sherman Labs., Detroit 11, Mich. *Dir. of Chem. Research.* (2R, 1912)

KOCH, ARTHUR L., Ph.D. U. of Florida, Dept. of Biochem., Gainesville. *Asst. Prof.* (2, 1956)

KOCHAKIAN, CHARLES D., Ph.D. U. of Alabama Med. Ctr., Dept. of Physiol., Birmingham 3. *Prof.* (1, 1942; 2, 1948)

KOCHER, RUDOLPH ALFRED, M.D. Box 936, Carmel, Calif. (2R, 1915)

KOCHOLATY, WALTER F., Ph.D. Army Med. Research Lab., Fort Knox, Ky. *Chief, Microbiol. Sec.* (2, 1950)

KOCSIS, JAMES J., Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Asst. Prof. in Pharmacol.* (3, 1957)

KOEHLER, ALFRED E., M.D., Ph.D. 225 Canon Dr, Santa Barbara, Calif. *Physician.* (2, 1924)

KOEHN, CARL J., Ph.D. Army Med. Research Lab., Fort Knox, Ky. (5, 1949)

KOEHNE, MARTHA, Ph.D. 1303 E. Main St., Columbus 5, Ohio. (5R, 1933)

KOELLA, WERNER PAUL, M.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. (1, 1954)

KOELLE, G. B., Ph.D., M.D. U. of Pa. Grad. Sch. Med., Philadelphia 4. *Prof. Pharmacol.; Chmn. Dept. of Physiol. & Pharmacol.; Dean.* (3, 1947)

KOENIG, VIRGIL L., Ph.D. Northwestern U. Med. Sch., Dept. of Biochem., Chicago 11, Ill. (2, 1953)

KOEPF, GEORGE F., M.D. 537 Delaware Ave., Buffalo 2, N. Y. *Assoc. in Med., U. of Buffalo.* (1, 1942)

KOEPPE, ROGER E., Ph.D. U. of Tennessee Sch. of Biol. Sci., Div. of Chem., Memphis 3. *Assoc. Prof.* (2, 1958)

KOERBER, WALTER L., Ph.D. Squibb Inst. for Med. Research, Georges Rd., New Brunswick, N. J. (6, 1943)

KOFFLER, HENRY, Ph.D. Purdue U., Dept. of Biol. Sci., West Lafayette, Ind. *Prof. Bacteriol.; Coordinator of Res. in Biol. Sci.* (2, 1958)

KOHLSTAEDT, KENNETH G., M.D. Eli Lilly & Co., Indianapolis 6, Ind. *Dir., Clin. Res.; Prof. of Med. Indiana U. Sch. of Med.* (1, 1947)

KOHN, HENRY I., Ph.D., M.D. U. of California Med. Ctr., Radiol., Lab., San Francisco 22. *Clin. Prof. Exptl. Radiol.* (1, 1940)

KOIZUMI, KIYOMI, M.D. State U. of New York Coll. of Med., Brooklyn 3. *Asst. Prof. of Physiol.* (1, 1957)

KOLETSKY, SIMON, M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Prof. of Pathol.* (4, 1951)

KOLFF, W. J., M.D. Cleveland Clinic, Research Dir., Cleveland, Ohio. *Member Staff; Dir., Dept. Artificial Organs.* (1, 1956)

KOLMER, JOHN A., M.D., D.P.H. 2101 Pine St., Philadelphia, Pa. *Prof. of Med., Temple U. and Dir., Inst. of Public Health & Prev. Med.* (6R, 1913)

KOMAROV, S. A., M.D., Ph.D. Temple U. Sch. of Med., Philadelphia 40, Pa. *Assoc. Prof. of Res. Physiol., Fels Res. Inst.* (1, 1933)

KOPELOFF, NICHOLAS, Ph.D. 23 Haven Ave., New York City 32. (6R, 1937)

KOPPANYI, THEODORE, Ph.D. Georgetown U. Sch. of Med., Washington 7, D. C. *Prof. of Pharmacol.*

(1, 1924; 3, 1935)

KOPROWSKI, HILARY, M.D. Wistar Inst., 36th & Woodland Ave., Philadelphia 4, Pa. *Dir.* (6, 1946)

KORN, EDWARD D., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Biochemist, Lab. of Cellular Physiol. and Metab.* (2, 1957)

KORNBERG, ARTHUR, M.D. Washington U. Sch. of Med., Dept. of Microbiol., St. Louis 10, Mo. *Prof. and Head of Dept.* (2, 1949)

KORNETSKY, CONAN, Ph.D. Natl. Inst. of Mental Health, Lab. of Clin. Sci., NIH, Bethesda 14, Md. *Research Psychologist.* (3, 1957)

KORNGOLD, LEONHARD, Ph.D. Sloan-Kettering Inst. for Cancer Research, New York City 21. (6, 1954)

KORR, IRVIN M., Ph.D. Kirksville Coll. of Osteopathy and Surgery, Kirksville, Mo. *Prof. of Physiol.* (1, 1939)

KORSON, ROY, M.D. U. of Vermont Coll. of Med., Burlington. *Assoc. Prof. of Pathol.* (4, 1956)

KOSHLAND, D. E., JR., Ph.D. Brookhaven Natl. Lab., Upton, N.Y. *Biochemist.* (2, 1954)

KOSHLAND, MARIAN ELLIOTT, Ph.D. Brookhaven Natl. Lab., Upton, N. Y. *Assoc. Bacteriologist.* (6, 1950)

KOSMAN, A. J., Ph.D. Northwestern U. Med. Sch., Dept. of Physiol., Chicago 11, Ill. *Assoc. Prof.* (1, 1949)

KOSTER, RUDOLF, Ph.D. Wellcome Research Labs., Tuckahoe 7, N. Y. *Pharmacologist.* (3, 1949)

KOTIN, PAUL, M.D. U. of Southern California Sch. of Med., Los Angeles 33. *Assoc. Prof. of Pathol.* (4, 1957)

KOTTKE, FREDERIC J., Ph.D., M.D. U. of Minnesota, Dept. of Phys. Med. & Rehabil., Minneapolis. *Prof. and Head of Dept.* (1, 1947)

KOZELKA, FRANK L., Ph.D. U. of Wisconsin, Univ. Hosp. and State Lab. of Hygiene, Madison 6. *Prof. of Clin. Pathol.* (3, 1939)

KOZLOFF, LLOYD M., Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Assoc Prof.* (2, 1951)

KRAATZ, CHARLES P., Ph.D. Jefferson Med. Coll., 1025 Walnut St., Philadelphia 3, Pa. *Prof. of Pharmacol.* (3, 1950)

KRAHL, MAURICE E., Ph.D. U. of Chicago, 951 E. 58th St., Chicago 37, Ill. *Prof. of Physiol.* (2, 1939; 3, 1949)

KRAKOWER, CECIL ALEXANDER, M.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Pathol.* (4, 1945)

KRALL, ALBERT R., Ph.D. R.I.A.S. 7212 Bellona Ave., Baltimore 12, Md. *Staff Biochemist.* (2, 1958)

KRAMAR, JENO, M.D. Creighton U. Sch. of Med., Omaha, Nebr. *Prof. of Pediatrics.* (1, 1958)

KRAMER, BENJAMIN, M.D. 4802-10th Ave., Brooklyn 19, N. Y. *Chief Pediatrician, Maimonides Hosp.; Clin. Prof., State U.* (1, 1915; 2, 1914)

KRAMER, MARTHA, Ph.D. Kansas State Coll., Manhattan. *Asst. Dean, Sch. of Home Econ.* (5, 1933)

KRAMPITZ, LESTER O., Ph.D. Western Reserve U., Cleveland 6, Ohio. *Prof. of Microbiol.; Dir. of Dept.* (2, 1946; 6, 1949)

KRANTZ, JOHN C., JR., Ph.D. U. of Maryland Med. Sch., Baltimore 1. *Prof. of Pharmacol.* (3, 1937)

KRATZER, F. H., Ph.D. U. of California, Dept. of Poultry Husbandry, Davis. *Prof.* (5, 1949)

KRAUSE, REGINALD F., Ph.D., M.D. West Virginia U. Sch. of Med., Dept. of Biochem., Morgantown. *Prof. and Head of Dept.* (2, 1955)

KRAUSS, WILLIAM E., Ph.D. Ohio Agric. Expt. Sta., Wooster. *Assoc. Dir.* (2, 1932; 5, 1933)

KRAYBILL, HERMAN F., Ph.D. U. S. Army Med. Nutrition Lab., Fitzsimons Army Hosp., Denver 8, Colo. *Chief, Chem. Div.* (5, 1956)

KRAYER, OTTO, M.D. Harvard Med. Sch., Boston 15, Mass. *Charles Wilder Prof of Pharmacol. and Head of Dept.* (3, 1938)

KREBS, EDWIN G., M.D. U. of Washington, Dept. of Biochem., Seattle 5. *Prof.* (2, 1952)

KREEZER, GEORGE L., Ph.D. Washington U., St. Louis 5, Mo. *Prof. of Psychol.* (1, 1948)

KREHL, WILLARD A., Ph.D., M.D. Yale U., New Haven 11, Conn. *Assoc. Prof. of Nutrition.* (2, 1947; 5, 1949)

KRIEGER, CARL H., Ph.D. Campbell Soup Co., Camden, N. J. *Dir., Basic Research and Product Develop.* (5, 1954)

KRIEGER, HOWARD P., M.D. Mt. Sinai Hosp., New York City 29. *Asst. Attending Neurologist.* (1, 1956)

KRITCHEVSKY, THEODORE H., Ph.D. 9010 S. Clyde St., Chicago 17, Ill. *Vice-Pres., Ninol Labs.* (2, 1952)

KRIVOY, WILLIAM A., Ph.D. Tulane U. Sch. of Med., Dept. of Pharmacol., New Orleans 12, La. *Instr.* (3, 1958)

KROC, ROBERT L., Ph.D. Warner-Lambert Research Inst., Morris Plains, N. J. *Dir. of Physiol. Research.* (1, 1957)

KROP, STEPHEN, Ph.D. Ethicon, Inc., Somerville, N. J. *Chief, Pharmacol. Dept.* (1, 1949; 3, 1944)

KRUEGER, ALBERT PAUL, M.D. U. of California, Berkeley. *Prof. Emer. of Bacteriol.; Lect. Emer. in Med.* (4, 1930; 6, 1937)

KRUEGER, HUGO, Ph.D. Oregon State Coll., Dept. of Dairy and Animal Husbandry, Corvallis. *Prof. of Physiol.* (1, 1931; 3, 1935)

KRUMBHAAR, EDWARD B., M.D., Ph.D. U. of Pennsylvania Med. Sch., Philadelphia. *Prof. Emer. of Pathol.* (1R, 1914; 4R, prior to 1920)

KRUSE, HARRY DAYTON, M.D., Sc.D. New York Acad. of Med., New York City 29. *Exec. Secy., Committee on Public Health.* (2, 1933; 5, 1950)

KRUSE, THEOPHILE K., Ph.D. 528 Overlook Dr., Pittsburgh 16, Pa. *Prof. Emer., Physiol. & Pharmacol., U. of Pittsburgh Med. Sch.* (1, 1919; 3R, 1920)

KUBICEK, WILLIAM G., Ph.D. U. of Minnesota Hosps., Dept. of Phys. Med. and Rehabil., Minneapolis. *Prof.* (1, 1947)

KUBIE, LAWRENCE S., M.D. 7½ E. 81st St., New York City 28. *Clin. Prof. of Psychiatry, Yale U.; Lect., Psychiatry, Columbia U.* (4, 1928)

KUBY, STEPHEN A., Ph.D. U. of Wisconsin, Inst. for Enzyme Research, Madison 6. *Asst. Prof.* (2, 1957)

KUFFLER, STEPHEN W., M.D. Johns Hopkins Med. Sch., Wilmer Inst., Baltimore 5, Md. *Prof. of Ophthal. Physiol. and Biophysics.* (1, 1949)

KUHN, HARRY A., M.S. Suite 900, Albee Bldg., 1426

G St., N. W., Washington 5, D. C. *Consultant.* (3, 1927)

KUHNS, WILLIAM J., M.D. U. of Pittsburgh Med. Ctr., Pittsburgh 13, Pa. *Assoc. Prof. of Pathol.; Dir., Central Blood Bank.* (6, 1954)

KUIKEN, KENNETH A., Ph.D. Buckeye Cellulose Corp., Cellulose and Specialties Tech. Div., Memphis 8, Tenn. (2, 1950)

KUIZENGA, MARVIN H., Ph.D. The Upjohn Co., Kalamazoo, Mich. *Asst. Dir., Biol. Research.* (2, 1947)

KULKA, JOHANNES P., M.D. Robert B. Brigham Hosp., Boston 20, Mass. *Clin. Assoc. in Pathol., Harvard Med. Sch.* (4, 1957)

KUMMEROW, FRED A., Ph.D. U. of Illinois, Dept. of Food Tech., Urbana. *Prof. of Food Chem.* (5, 1957)

KUN, ERNEST, M.D. U. of California Sch. of Med., Dept. of Pharmacol., San Francisco 22. *Estab. Invest., Amer. Heart Assn.* (2, 1954; 3, 1949)

KUNDE, MARGARETE M., Ph.D., M.D. 30 N. Michigan Ave., Chicago, Ill. *Instr. in Med., Northwestern U.; Staff, Wesley Mem. Hosp.* (1R, 1925)

KUNITZ, MOSES, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (2R, 1947)

KUNKEL, HARRIOTT O., Ph.D. Texas A. & M. Coll., Dept. of Biochem. and Nutrition, College Station. *Prof.* (2, 1954)

KUNKEL, HENRY G., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (2, 1953)

KUPFER, SHERMAN, M.D. 1176 Fifth Ave., New York City 29. *Asst. to Dir. Med. Res., Mt. Sinai Hosp.; Asst. Prof. Physiol., Cornell U.* (1, 1958)

KUPKE, D. W., Ph.D. U. of Virginia Sch. of Med., Charlottesville. *Asst. Prof. of Biochem.* (2, 1957)

KUPPERMAN, HERBERT S., M.D., Ph.D. New York U. Postgrad. Med. Sch., Dept. of Therap., New York City 16. *Assoc. Prof. of Med.* (3, 1950)

KURTZ, ALTON C., Ph.D. U. of Oklahoma Med. Sch., Dept. of Biochem., Oklahoma City 4. *Prof.* (2, 1942)

KUSCHNER, MARVIN, M.D. New York U. Coll. of Med., New York City 16. *Prof. of Pathol.; Dir. Pathol., Bellevue Hosp.* (4, 1956)

KUYPER, ADRIAN C., Ph.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Assoc. Prof. of Physiol. Chem.* (2, 1946)

KWIT, NATHANIEL T., M.D. New York U. Coll. of Dent., Dept. of Pharmacol., New York City 10. *Asst. Prof.* (3, 1955)

KYDD, D. M., M.D. State U. of New York Coll. of Med., Dept. of Int. Med., Brooklyn 3. *Prof. of Med.* (5, 1934)

KYKER, GRANVIL C., Ph.D. Oak Ridge Inst. of Nuclear Studies, Med. Div., P. O. Box 117, Oak Ridge, Tenn. *Chief Scientist.* (2, 1947)

LA BELLE, ANNETTE, B.A. 218 W. Thomas St., Rome, N. Y. (1, 1948)

LACKEY, ROBERT W., Ph.D. U. of Texas, Southwestern Med. Sch., Dept. of Physiol., Dallas 19. *Prof. and Chmn. of Dept.* (1, 1947)

LACKMAN, DAVID B., Ph.D. USPHS, Rocky Mountain Lab., Hamilton, Mont. *Chief, Serol.-Pathol. Sec.* (6, 1952)

LACORTE, JOSE G., M.D. Instituto Oswaldo Cruz, Rio de Janeiro, Brazil. *Chief, Virus Sec.; Prof., U. of Brazil.* (6, 1946)

LACY, G. R., M.D. 1364 Navahoe Dr., Pittsburgh 28, Pa. (4R, 1927)

LACY, PAUL E., Ph.D., M.D. Washington U. Sch. of Med., Dept. of Pathol., St. Louis 10, Mo. *Asst. Prof.* (4, 1957)

LA DU, BERT N., JR., M.D., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Clin. Invest.* (2, 1956)

LAIRD, ANNA KANE, M.D., Ph.D. Argonne Natl. Lab., P. O. Box 299, Lemont, Ill. *Assoc. Biologist.* (4, 1954)

LAJTHA, ABEL, Ph.D. New York State Psychiatric Inst., New York City 32. *Assoc. Res. Scientist; Asst. Prof. Biochem., Columbia U.* (2, 1958)

LALICH, JOSEPH J., M.D. U. of Wisconsin, 426 N. Charter St., Madison 6. *Prof. of Pathol.* (4, 1946)

LAMB, ALVIN R., Ph.D. 60 Mariposa Ave., Los Gatos, Calif. (2R, 1923; 5, 1934)

LAMBERT, EDWARD H., Ph.D., M.D. Mayo Fndn., Rochester, Minn. *Assoc. Prof. of Physiol.* (1, 1945)

LAMBERT, ROBERT A., M.D. Box 54, Fairhope, Ala. (4, 1922)

LAMBERTSEN, CHRISTIAN J., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Pharmacol.* (1, 1955; 3, 1948)

LAMBOOY, JOHN P., Ph.D. U. of Rochester Sch. of Med. and Dent., Dept. of Physiol., Rochester 20, N. Y. *Assoc. Prof.* (2, 1953)

LAMPEN, J. OLIVER, Ph.D. Squibb Inst. for Med. Research, New Brunswick, N. J. *Dir., Div. of Biochem. Research.* (2, 1947)

LAMPORT, HAROLD, M.D. Yale U. Sch. of Med., New Haven, Conn. *Research Assoc. in Physiol.* (1, 1943)

LAMSON, BALDWIN G., M.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Pathol.* (4, 1952)

LAMSON, PAUL DUDLEY, M.D. Vanderbilt U. Sch. of Med., Nashville, Tenn. *Prof. Emer. of Pharmacol.* (1R, 1921; 3R, 1915)

LANCEFIELD, REBECCA C., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (6, 1933)

LANDAU, WILLIAM M., M.D. Washington U. Med. Sch., St. Louis 10, Mo. *Assoc. Prof. of Neurol.* (1, 1955)

LANDING, B. H., M.D. Children's Hosp. Res. Fndn., Cincinnati 29, Ohio. *Pathologist; Asst. Prof. Pathol. & Pediat., U. Cincinnati.* (4, 1957)

LANDIS, CARNEY, Ph.D. Columbia U. Psychiatric Inst., 722 W. 168th St., New York City. *Prin. Research Psychologist; Prof. of Psychol.* (1, 1939)

LANDIS EUGENE MARKLEY, Ph.D., M.D. Harvard Med. Sch., Boston 15, Mass. *George Higginson Prof. of Physiol.* (1, 1928)

LANDOWNE, MILTON, M.D. Levindale, Baltimore 15, Md. *Med. Dir.* (1, 1947)

LANDS, ALONZO M., Ph.D. Sterling-Winthrop Res. Inst., Rensselaer, N. Y. *Asst. Dir., Biol. Div.; Head, Pharmacol. Sec.* (1, 1942; 3, 1947)

LANDY, MAURICE, Ph.D. Natl. Cancer Inst., Lab. of Chem. Pharmacol., NIH, Bethesda 14, Md.

Head, Polysaccharide Sec. (6, 1952)

LANE, ARDELLE C., Ph.D. Northwestern U. Dental Sch., Chicago 11, Ill. *Asst. Prof. of Physiol. and Pharmacol.* (1, 1958)

LANE, CHARLES E., Ph.D. U. of Miami, Marine Lab., Virginia Key, Miami 49, Fla. *Assoc. Prof. of Marine Sci.* (1, 1952)

LANGDELL, ROBERT D., M.D. U. of North Carolina, Dept. of Pathol., Chapel Hill. *Asst. Prof.* (4, 1957)

LANGDON, ROBERT G., Ph.D., M.D. Johns Hopkins Sch. of Med., Dept. of Physiol. Chem., Baltimore 5, Md. *Asst. Prof.* (2, 1956)

LANGE, KURT, M.D. New York Med. Coll., Metropolitan Med. Ctr., New York City 29. *Assoc. Prof., Med.; Assoc. Prof., Pediatrics (Res.).* (4, 1957)

LANGEMANN, HEINRICH, M.D. Pharmakologisches Inst., U. of Zurich, Zurich, Switzerland. *Asst.* (3, 1953)

LANGHAM, WRIGHT H., Ph.D. U. of California, Los Alamos Sci. Lab., P. O. Box 1663, Los Alamos, N. Mex. *Leader, Biomed. Res. Group.* (2, 1951)

LANGLEY, L. L., Ph.D., LL.B. Med. Coll. of Alabama, Birmingham 5. *Assoc. Prof. of Physiol.* (1, 1951)

LANGLEY, WILSON D., Ph.D. U. of Buffalo Med. Sch., Dept. of Biochem., Buffalo, N. Y. *Prof. and Head of Dept.* (2, 1937)

LANNI, FRANK, Ph.D. Emory U., Dept. of Bacteriol., Atlanta 22, Ga. (6, 1949)

LANPHIER, EDWARD H., M.D. USN Underwater Demolition Team #21, USN Amphibious Base, Norfolk, Va. *Med. Officer.* (1, 1956)

LAQUEUR, GERT LUDWIG, M.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. (4, 1952)

LARDY, HENRY A., Ph.D. U. of Wisconsin, Inst. for Enzyme Research, Madison 6. *Prof. of Biochem.* (2, 1946)

LARNER, JOSEPH, M.D., Ph.D. Western Reserve U. Sch. of Med., Dept. of Pharmacol., Cleveland 6, Ohio. *Assoc. Prof.* (2, 1953)

LARRABEE, MARTIN G., Ph.D. Johns Hopkins U., Biophysics Dept., Baltimore 18, Md. *Assoc. Prof.* (1, 1940)

LARSEN, ELEANOR M., Ph.D. U. of Wisconsin, Serv. Mem. Insts., Dept. of Physiol., Madison 6. *Asst. Prof.* (1, 1949)

LARSON, BRUCE L., Ph.D. U. of Illinois, Urbana. *Asst. Prof. of Dairy Biochem.* (2, 1957)

LARSON, CARL L., M.D. Rocky Mountain Lab., Natl. Inst. of Allergy & Infectious Diseases, NIH, Hamilton, Mont. *Dir.* (6, 1948)

LARSON, DANIEL L., M.D. Columbia U. Coll. of P. & S., New York City 32. *Asst. Prof. of Med.* (6, 1953)

LARSON, EDWARD, Ph.D. U. of Miami, Dept. of Zool., Coral Gables 46, Fla. *Prof. of Physiol. and Zool.* (1, 1929; 3, 1937)

LARSON, HARDY W., Ph.D. Metropolitan Life Insurance Co., Biochem. Lab., 1 Madison Ave., New York City. *Research Chemist.* (2, 1937)

LARSON, PAUL S., Ph.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Pharmacol.* (1, 1939; 3, 1947)

LASAGNA, LOUIS, M.D. Johns Hopkins Sch. of Med., Depts. of Pharmacol. and Med., Baltimore 5, Md. (3, 1953)

LASHLEY, K. S., Ph.D. Yerkes Labs., Orange Park, Fla. *Dir., Emer.; Res. Prof. Emer. Neuropsychol., Harvard.* (1R, 1923)

LASKOWSKI, M., Ph.D. Marquette U. Med. Sch., Milwaukee 3, Wis. *Prof. of Biochem.* (2, 1944)

LASKOWSKI, M., JR., Ph.D. Purdue U., Dept. of Chem., Lafayette, Ind., *Asst. Prof.* (2, 1957)

LAST, JULES H., Ph.D., M.D. 2360 Woodpath, Highland Park, Ill. *Asst. Prof. of Med., Northwestern U. Med. Sch., Chicago.* (3, 1948)

LATTA, HARRISON, M.D. U. of California Sch. of Med., Los Angeles. *Assoc. Prof. of Pathol.* (4, 1954)

LAUFFER, MAX A., JR., Ph.D. U. of Pittsburgh, Pittsburgh 13, Pa. *Dean, Div. of Natural Sci. and Prof. of Biophysics.* (2, 1946)

LAUG, E. P., Ph.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D. C. *Chief, Physio-Chem. Br.* (2, 1938; 3, 1947)

LAURENS, HENRY, Ph.D. Box 157, Flatrock, N. C. (1R, 1913)

LAUSON, HENRY D., Ph.D., M.D. Albert Einstein Coll. of Med., Dept. of Physiol., New York City 61. *Prof. and Chmn. of Dept.* (1, 1946)

LAVIK, PAUL S., Ph.D. Western Reserve U., Cleveland 6, Ohio. *Assoc. Prof. of Biochem.; Asst. Prof. of Radiol.* (2, 1952)

LAVINE, T. F., Ph.D. Inst. for Cancer Research, Fox Chase, Philadelphia 11, Pa. *Sr. Member.* (2, 1938)

LAW, LLOYD WILLIAM, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Leukemia Studies Sec.* (4, 1952)

LAWRENCE, H. SHERWOOD, M.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (6, 1951)

LAWRENCE, JOHN H., M.D. U. of California, Donner Lab., Berkeley 4. *Dir.; Prof. Med. Physics; Physician-in-Chief, Donner Pav.* (1, 1952)

LAWRENCE, MERLE, Ph.D. U. of Michigan Med. Sch., Dept. of Otolaryngol., Ann Arbor. *Prof. of Physiol. Acoustics.* (1, 1956)

LAWRENCE, W. SHERWOOD, M.D. 405 Sycamore St., Gridley, Calif. *Physician and Surgeon.* (3, 1944)

LAWSON, HAMPDEN C., M.D., Ph.D. U. of Louisville, Louisville, Ky. *Prof. of Physiol.* (1, 1933)

LAWTON, ALFRED H., M.D., Ph.D. 16438 Redington Dr., St. Petersburg, Fla. *Dir., Res. Lab. & Ward Physician, VA Ctr., Bay Pines.* (1, 1949; 3, 1948)

LAWTON, RICHARD W., M.D. Aviation Med. Acceleration Lab., Naval Air Develop. Ctr., Johnsville, Pa. *Head, Physiol. Div.* (1, 1951)

LAZARUS, SYDNEY S., M.D. Isaac Albert Res. Inst., Brooklyn 3, N. Y. *Chief of Pathol.* (4, 1958)

LEAKE, CHAUNCEY D., Ph.D. Ohio State U., Hamilton Hall, Columbus 10. *Prof. of Pharmacol.* (1, 1923; 3, 1924)

LEASE, ELMER J., Ph.D. Clemson Agric. Coll., Dept. of Nutrition, Clemson, S. C. *Head.* (5, 1958)

LEATHEM, JAMES H., Ph.D. Rutgers, The State U., New Brunswick, N. J. *Prof. of Zool.* (1, 1945)

LeBLANC, JACQUES A., Ph.D. Laval U. Med. Sch.,

Dept. of Physiol., Quebec, Canada. (1, 1957)

LEDER, IRWIN G., Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Biochemist.* (2, 1957)

LEDERER, L. G., Ph.D., M.D. Natl. Airport, Washington, D. C. *Med. Dir., Capital Airlines; Asst. Clin. Prof. Med. Geo. Washington U.* (1, 1940)

LEDUC, ELIZABETH H., Ph.D. Brown U., Providence 12, R. I. *Assoc. Prof. of Biol.* (4, 1958)

LEE, CHENG-CHUN, Ph.D. Lilly Research Labs., Indianapolis 6, Ind. *Pharmacologist.* (1, 1956; 3, 1957)

LEE, DOUGLAS H. K., M.D. QM Research & Engin. Command, Natick, Mass. *Asst. Sci. Dir. for Research.* (1, 1949)

LEE, HENRY M., Ph.D. Eli Lilly & Co., 740 South Alabama, Indianapolis 6, Ind. *Pharmacologist.* (3, 1952)

LEE, KWANG SOO, Ph.D., M.D. State U. of New York Coll. of Med., Dept. of Pharmacol., Brooklyn 3. *Assoc. Prof.* (3, 1953)

LEE, MILTON O., Ph.D. 9650 Wisconsin Ave., Washington 14, D. C. *Managing Editor, Amer. Physiol. Soc.; Federation Secy.* (1, 1927; 5, 1933)

LEE, N. D., Ph.D. VA Med. Teaching Group Hosp., Radioisotope Serv., Memphis, Tenn. *Asst. Chief; Asst. Prof. Chem., U. of Tenn. Med. Sch.* (2, 1955)

LEE, RICHARD E., Ph.D., M.D. New York Hosp., Dept. of Med., New York City 21. *Asst. Prof. of Med., Cornell Med. Coll.* (1, 1950)

LEESE, CHESTER E., Ph.D. George Washington U. Sch. of Med., Washington 5, D. C. *Prof. of Physiol.* (1, 1934)

LeFEVRE, PAUL G., Ph.D. Brookhaven Natl. Lab., Upton, N. Y. *Scientist, Med. Dept.* (1, 1950)

LEHMAN, ARNOLD J., Ph.D., M.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D. C. *Dir.* (3, 1937)

LEHMAN, ROBERT A., Ph.D. 254 W. 31st St., New York City 1. *Dir. of Research, Campbell Pharmaceutical Co.* (3, 1942)

LEHNINGER, ALBERT L., Ph.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *De Lamar Prof. of Physiol. Chem.; Dir. of Dept.* (2, 1946; 5, 1954)

LEHR, DAVID, M.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City 29. *Prof. & Dir., Dept. Physiol. & Pharmacol.* (3, 1947)

LEICHSENRING, JANE M., Ph.D. U. of Minnesota, Sch. of Home Econ., St. Paul. *Prof. of Nutrition.* (5, 1948)

LEIGHTON, JOSEPH, M.D. U. of Pittsburgh Sch. of Med., Dept. of Pathol., Pittsburgh 13, Pa. (4, 1954)

LEIN, ALLEN, Ph.D. Northwestern U. Med. Sch., Dept. of Physiol., Chicago 11, Ill. *Assoc. Prof.* (1, 1946)

LELOIR, LUIS F., M.D. Instituto de Investigaciones Bioquimicas, Obligado 2490, Buenos Aires, Argentina. *Dir.* (2, 1957)

LENNETTE, EDWIN H., Ph.D., M.D. California State Dept. of Public Health, Berkeley 4. *Chief, Viral & Rickettsial Dis. Lab.* (4, 1941; 6, 1947)

LEON, MYRON, Ph.D. St. Luke's Hosp., 11311 Shaker Blvd., Cleveland 4, Ohio. *Assoc. in Surgical Res.* (6, 1958)

LEONARD, CLIFFORD SHATTUCK, Ph.D. 286 Whitfield St., Guilford, Conn. *Tech. Assoc., Chem.-Biol. Coord. Ctr., Natl. Research Council.* (3, 1927)

LEONARDS, JACK RALPH, Ph.D., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof. of Biochem.* (2, 1948)

LEONE, CHARLES A., Ph.D. U. of Kansas, Dept. of Zool., Lawrence. (6, 1954)

LE PAGE, G.A., Ph.D. U. of Wisconsin, McArdle Mem. Lab. for Cancer Research, Madison 6. *Prof. of Oncol.* (2, 1949)

LEPESCHKIN, EUGENE, M.D. U. of Vermont Coll. of Med., Burlington. *Assoc. Prof. of Exptl. Med.* (1, 1955)

LEPKOVSKY, SAMUEL, Ph.D. U. of California, Poultry Dept., Berkeley 4. *Prof. of Poultry Husbandry.* (2, 1933; 5, 1933)

LEPOW, IRWIN H., Ph.D., M.D. Western Reserve U. Inst. of Pathol., Cleveland 6, Ohio. *Asst. Prof. of Exptl. Pathol.* (6, 1954)

LERNER, AARON B., M.D., Ph.D. Yale U. Sch. of Med., Dept. of Med., New Haven 11, Conn. *Prof.; Head, Sec. of Dermatol.* (2, 1952)

LERNER, EDWIN M., II, M.D. Natl. Inst. Arthritis & Metabolic Diseases, Lab. Pathol. & Histochem., NIH, Bethesda 14, Md. *Pathologist.* (4, 1956)

LESKOWITZ, SIDNEY, Ph.D. Harvard Med. Sch., Boston 15. Mass. *Res. Assoc., Bacteriol. & Immunol.; Assoc. Immunologist, Mass. Gen. Hosp.* (6, 1956)

L'ESPERANCE, ELISE L., M.D. 535 Pelham Manor Rd., Pelham Manor, N. Y. (6, 1920)

LESSLER, MILTON A., Ph.D. Ohio State U. Coll. of Med., Columbus 10. *Assoc. Prof. of Physiol.* (1, 1952)

LETTVIN, JEROME Y., M.D. Massachusetts Inst. of Tech., Cambridge 39. *Research Assoc.* (1, 1955)

LEUCHTENBERGER, CECILIE, Ph.D. Western Reserve U., Inst. of Pathol., Cleveland 6, Ohio. *Assoc. Prof. of Cytol. and Biol.* (4, 1954)

LEVENBOOK, L., Ph.D. Natl. Inst. Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Biochemist.* (1, 1955)

LEVERTON, RUTH M., Ph.D. USDA, Inst. of Home Econ., ARS, Washington 25, D. C. *Assoc. Dir., Human Nutrition Res. Div.* (5, 1942)

LEVEY, STANLEY, Ph.D. University Hosps., Cleveland 6, Ohio. *Asst. Prof. Biochem., Western Reserve U.* (2, 1951; 5, 1958)

LEVIN, LOUIS, Ph.D. Natl. Science Fndn., Washington 25, D. C. *Prog. Dir., Metabolic Biol.; Dep. Asst. Dir., Biol. and Med. Sci.* (2, 1939)

LEVINE, HAROLD, Ph.D. 941 E. Sylvan Ave., Milwaukee 17, Wis. *Harmel Products.* (2, 1933; 5, 1933)

LEVINE, LAWRENCE, Sc.D. Brandeis U., Waltham, Mass. *Asst. Prof. of Biochem.* (6, 1956)

LEVINE, PHILIP, M.D. Ortho Research Fndn., Raritan, N. J. *Dir. of Immunohematol.* (6, 1925)

LEVINE, RACHMIEL, M.D. Michael Reese Hosp., Chicago, Ill. *Chmn., Dept. of Med.* (1, 1942)

LEVINE, RUTH R., Ph.D. Tufts U. Sch. of Med., Boston 11, Mass. *Asst. Prof. of Pharmacol.* (3, 1956)

LEVINE, SAMUEL Z., M.D. 525 E. 68th St., New York City. *Prof. of Pediatrics, Cornell U.; Pediatrician-in-Chief, New York Hosp.* (5, 1933)

LEVINE, VICTOR EMANUEL, Ph.D., M.D. Creighton U. Sch. of Med., Omaha, Nebr. *Prof. of Biol. Chem. and Nutrition.* (2, 1936)

LEVINSON, SAMUEL A., Ph.D., M.D. U. of Illinois Coll. of Med., Chicago 12. *Prof. of Pathol.* (4, 1938)

LEVINTOW, LEON, M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Surgeon, PHS.* (2, 1954)

LEVITT, MARVIN F., M.D. The Mount Sinai Hosp., 5th Ave. and 100th St., New York City 29. *Research Assoc., Dept. of Med.* (1, 1953)

LEVITZ, MORTIMER, Ph.D. New York U. Coll. of Med., New York City 16. *Asst. Prof. Res. Obstetrics & Gynecol. (Chem.).* (2, 1958)

LEVY, BARNET M., D.D.S. U. of Texas Dental Br., Houston 25. *Prof. of Pathol.* (4, 1951)

LEVY, MATTHEW NATHAN, M.D. St. Vincent Charity Hosp., Cleveland 15, Ohio. *Dir., Research Dept.,* (1, 1951)

LEVY, MILTON, Ph.D. New York U. Coll. of Dent., New York City 10. *Prof. and Chmn., Dept. of Biochem.* (2, 1933)

LEVY, ROBERT L., M.D. 730 Park Ave., New York City 21. *Prof. Emer. Clin. Med., Columbia U., Coll. of P. & S.* (3, 1915)

LEWIS, ALVIN EDWARD, M.D. Mt. Zion Hosp., 1600 Divisadero St., San Francisco 15, Calif. *Pathologist.* (1, 1953)

LEWIS, BENJAMIN M., M.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Asst. Prof. of Med.* (1, 1957)

LEWIS, DAVID HAROLD, M.D. U. of Pennsylvania Grad. Sch. of Med., Philadelphia 4. *Assoc. in Cardiol.* (1, 1957)

LEWIS, GEORGE T., Ph.D. U. of Miami Sch. of Med., Dept. of Biochem., Coral Gables 34, Fla. *Prof. and Chmn. of Dept.* (2, 1949)

LEWIS, GLADYS KINSMAN, Ph.D. 444 Fairfax, Denver 20, Colo. *Asst. Prof., Prev. Med. and Public Health, U. of Colo. Sch. of Med.* (5, 1944)

LEWIS, JAMES C., Ph.D. USDA, Western Utilization Research Br., Albany 10, Calif. *Unit Leader.* (2, 1946)

LEWIS, JESSICA H., M.D. U. of Pittsburgh, Dept. of Med., Pittsburgh 13, Pa. *Research Assoc.* (1, 1949)

LEWIS, JOHN R., Ph.D. Sterling-Winthrop Research Inst., Rensselaer, N. Y. *Asst. Head of Sec. of Coordination.* (3, 1950)

LEWIS, J. H., M.D. 4750 Champlain Ave., Chicago, Ill. *Assoc. Prof. Pathol., U. of Chicago; Member, Otho S. A. Sprague Mem. Inst.* (4, 1924)

LEWIS, LENA A., Ph.D. Cleveland Clinic, Cleveland 6, Ohio. *Res. Staff Member, Cleveland Clinic Fndn.* (1, 1946)

LEWIS, ROBERT C., Ph.D. 444 Fairfax, Denver 20, Colo. *Prof. Emer. of Biochem., U. of Colo. Sch. of Med.* (2R, 1931; 5R, 1933)

LEWIS, URBAN J., Ph.D. Merck and Co., Inc., Rahway, N. J. *Sr. Biochemist.* (2, 1956)

LEWIS, WARREN H., M.D. Wistar Inst. of Anatomy and Biol., Philadelphia, Pa. *Member.* (1R, 1919)

LEY, HERBERT L., JR., M.D. 8911 Ewing Dr., Bethesda 14, Md. *Prof. of Bacteriol., George Washington U. Sch. of Med.* (6, 1949)

L'HEUREUX, MAURICE V., Ph.D. Loyola U., Stritch Sch. of Med., Chicago 12, Ill. *Assoc. Prof. of Biochem.* (2, 1958)

LI, CHEN PIEN, M.D. Div. of Biologics Standards, NIH, Bethesda 14, Md. *Bacteriologist.* (6, 1956)

LI, CHOH HAO, Ph.D. U. of California, 4596 Life Sci. Bldg., Berkeley. *Prof. of Biochem. & Exptl. Endocrinol.; Dir., Hormone Res. Lab.* (2, 1944)

LI, CHOH-LUH, M.D., Ph.D. Natl. Inst. of Neurological Diseases and Blindness, NIH, Bethesda 14, Md. *Assoc. Neurosurgeon.* (1, 1956)

LI, TSUNG HAN, M.D., D.Sc. (Med.) New England Med. Ctr., Boston 11, Mass. *Asst. Prof., Tufts Med. Sch.* (3, 1951)

LIAO, SUNG J., M.D. Waterbury Hosp., Waterbury, Conn. *Dir., Physical Med. and Rehabil.* (6, 1954)

LIBBY, RAYMOND L., Ph.D. U. of California Med. Ctr., Los Angeles 24. *Prof. of Radiol.* (6, 1938)

LIBERSON, W. T., M.D. Rocky Hill VA Hosp., Rocky Hill, Conn. *Neurophysiologist.* (1, 1948)

LIBET, BENJAMIN, Ph.D. U. of California Med. Ctr., 762 Med. Sci. Bldg., San Francisco 22. *Assoc. Prof. of Physiol.* (1, 1942)

LICHSTEIN, HERMAN C., Sc.D. U. of Minnesota Med. Sch., Dept. of Bacteriol. and Immunol., Minneapolis 14. *Prof.* (2, 1952)

LICKLIDER, J. C. R., Ph.D. Bolt Beranek and Newman Inc., Cambridge, Mass. *Dept. of Engin. Psychol.* (1, 1938)

LIDDELL, HOWARD S., Ph.D. Cornell U., Ithaca, N. Y. *Prof. of Psychobiol.; Dir. of Behavior Farm Lab.* (1, 1925)

LIEBERMAN, ARNOLD L., M.D. Ph.D. 41-42 Elbertson Street, Elmhurst 73, N. Y. (1, 1934)

LIEBERMAN, IRVING, D.V.M., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Microbiol., Pittsburgh 13, Pa. *Assoc. Prof.* (2, 1955)

LIEBERMAN, SEYMOUR, Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Biochem.* (2, 1948)

LIEF, PHILIP A., M.D. Mt. Sinai Hosp., Dept. of Anesthesiol., 1 E. 100th St., New York City 29. *Anesthesiologist.* (3, 1952)

LIENER, IRVIN E., Ph.D. U. of Minnesota, Dept. of Agric. Biochem., St. Paul 8. *Assoc. Prof.* (2, 1953; 5, 1955)

LIFSON, NATHAN, M.D., Ph.D. 2602 W. 54th St., Minneapolis, Minn. *Prof. of Physiol., U. of Minnesota Med. Sch.* (1, 1944)

LIGHTBODY, HOWARD D., Ph.D. R.F.D. 3, Box 17-B, Mena, Ark. *U. S. Civil Serv., Retired.* (2, 1936)

LIGON, EDGAR WILLIAM, JR., Ph.D. USDA, ARS, Pesticide Regulation Sec., Washington 25, D. C. *Chief, Pharmacol. and Rodenticide Unit.* (3, 1948)

LILJESTRAND, GORAN, M.D. Ph.D. Caroline Inst., Stockholm, Sweden. *Emer. Prof. of Pharmacol.; Secy. Nobel Comm. for Physiol. and Med.* (1H, 1950)

LILLIE, R. D., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Lab. of Pathol. and Histochem.; Med. Dir., PHS.* (4, 1941)

LILLIE, ROBERT J., Ph.D. USDA, Animal and Poultry Husbandry Research Br., Beltsville, Md. *Poultry Husbandman.* (5, 1956)

LILLY, JOHN C., M.D. Natl. Inst. of Mental Health, NIH, Bethesda 14, Md. *Chief, Sec. on Cortical*

Integration, Lab. of Neurophysiol. (1, 1950)

LIM, ROBERT K. S., M.B., Ph.D., D.Sc. Miles-Ames Research Lab., Elkhart, Ind. (1, 1923; 3, 1952)

LIN, TSUNG-MIN, Ph.D. Lilly Research Labs., Eli Lilly & Co., Indianapolis 6, Ind. *Pharmacologist.* (1, 1956)

LINDBERG, ROBERT B., Ph.D. USAREUR Medical Lab., Landstuhl Army Med. Ctr., Dept. of Microbiol., APO 180, New York City. *Chief.* (6, 1956)

LINDEMAN, V. F., Ph.D. Syracuse U., 16 Lyman Hall, Syracuse, N.Y. *Prof. of Zool.* (1, 1949)

LINDENBAUM, ARTHUR, Ph.D. Argonne Natl. Lab., Div. of Biol. and Med. Research, Lemont, Ill. *Assoc. Biochemist.* (2, 1956)

LINDSAY, STUART, M.D. U. of California Sch. of Med., H. C. Moffitt Hosp., San Francisco 22. *Prof. and Patbologist.* (4, 1949)

LINDSLEY, DONALD B., Ph.D. U. of California, Dept. of Psychol., Los Angeles 24. *Prof. of Psycbol. and Pbysiol.* (1, 1937)

LINEGAR, CHARLES R., Ph.D. E. R. Squibb & Sons New Brunswick, N. J. *Assoc. Dir., Pharmacol. Div.* (3, 1938)

LINEWEAVER, HANS, Ph.D. USDA Western Utilization Research and Develop. Div., Albany 10, Calif. *Chief, Poultry Lab.* (2, 1941)

LING, GILBERT N., Ph.D. Eastern Pennsylvania Psychiatric Inst., Philadelphia 29. *Sr. Research Scientist.* (1, 1951)

LINK, KARL PAUL, Ph.D. U. of Wisconsin, Biochem. Bldg., Madison. *Prof. of Biochem.* (2, 1931)

LINKER, ALFRED, Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Research Assoc.* (2, 1956)

LINTZ, WILLIAM, M.D. 36 Plaza St., Brooklyn, N.Y. *Late Prof. of Immunol. and Bacteriol.; Clin. Prof. of Med., Long Island Coll. of Med.* (6R, 1920)

LIONETTI, FABIAN J., Ph.D. Boston U. Sch. of Med., Dept. of Biochem., Boston, Mass. *Assoc. Prof.* (2, 1958)

LIPKIN, MARTIN, M.D. Bellevue Hosp., 2nd (Cornell) Med. Div., New York City. *Dir., Gastrointestinal Labs., Instr., Dept. Med., Cornell U.* (1, 1957)

LIPMANN, FRITZ, M.D., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1941)

LIPPINCOTT, STUART W., M.D. Brookhaven Natl. Lab., Upton, N.Y. *Sr. Scientist and Asst. to the Dir., Med. Dept.* (4, 1947)

LIPPMAN, RICHARD W., M.D. California Inst. of Technology, Pasadena. *Med. Research Consultant, Div. of Chem. and Chem. Engin.* (1, 1950)

LIPTON, MORRIS A., Ph.D., M.D. VA Res. Hosp., Chicago 11, Ill. *Chief, Investigative Med. Serv.; Asst. Prof. Med., Northwestern U.* (2, 1946)

LIPTON, MURRAY M., Ph.D. U. of Louisville Sch. of Med., Louisville 2, Ky. *Asst. Prof. of Child Health.* (6, 1954)

LISCO, HERMANN, M.D. Argonne Natl. Lab., Lemont, Ill., and Dept. of Pathol., U. of Chicago. (4, 1947)

LITCHFIELD, JOHN T., JR., M.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Dir., Exptl. Therap. Sec.* (3, 1940)

LITTLE, HENRY N., Ph.D. U. of Massachusetts, Dept. of Chem., Amherst. *Prof.* (2, 1958)

LITTLE, JAMES MAXWELL, Ph.D. Bowman Gray Sch. of Med., Winston-Salem 7, N.C. *Prof. Pharmacol.; Assoc. Prof. Physiol.* (1, 1942; 3, 1947)

LITTLE, ROBERT C., M.D. Seton Hall Coll. of Med., Dept. of Physiol., Jersey City 4, N.J. *Prof.* (1, 1950)

LITWACK, GERALD, Ph.D. Rutgers, The State U., New Brunswick, N.J. *Assoc. Prof. of Biochem.* (2, 1957)

LIU, CH'IEN, M.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. in Bacteriol. and Immunol.* (6, 1953)

LIU, OSCAR C., M.D., D.M.Sc. Merck Sharp and Dohme Research Labs., Merck & Co., Inc., West Point, Pa. (6, 1957)

LIVE, ISRAEL, V.M.D., Ph.D. U. of Pennsylvania Sch. of Veterinary Med., Philadelphia 4, Pa. *Prof. of Microbiol.* (6, 1955)

LIVINGSTON, ALFRED E., Ph.D. St. John's U., Jamaica 32, N.Y. *Coordinator of Sci. Labs.* (1R, 1917; 3R, 1920)

LIVINGSTON, ROBERT B., M.D. Natl Insts. of Health, Bethesda 14, Md. *Dir. of Basic Research, NIMH-NINDB.* (1, 1949)

LLOYD, CHARLES W., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Prof., Obstetrics; Assoc. Prof. Med.* (1, 1955)

LLOYD, DAVID P. C., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (1, 1939)

LLOYD, LEWIS E., Ph.D. Macdonald Coll., Dept. of Nutrition, Macdonald College, P. Q., Canada. *Assoc. Prof.* (5, 1958)

LOCKE, ARTHUR P., Ph.D. 1730 Morrill Street, Sarasota, Fla. (6R, 1926)

LODHOLZ, EDWARD, M.D. U. of Pennsylvania, Med. Labs., Philadelphia 4. *Prof. Emer. of Physiol., Grad. Sch. of Med.* (1R, 1913)

LOEB, LEO, M.D. 40 Crestwood Dr., St. Louis 5, Mo. *Prof. Emer. of Pathol., Washington U. Sch. of Med.* (1R, 1907; 4, 1913)

LOEBEL, ROBERT O., M.D. 205 East 78th St., New York City. (1R, 1928)

LOEFER, JOHN B., Ph.D. Office of Naval Research, 1030 E. Green St., Pasadena 1, Calif. *Coordinator for Biol. Sci.* (1, 1949)

LOEW, EARL R., Ph.D. Boston U. Sch. of Med., Dept. of Physiol., Boston 18, Mass. *Prof. and Head of Dept.* (1, 1940; 3, 1946)

LOEWE, LEO, M.D. 177 New York Ave., Brooklyn 16, N.Y. (3, 1951)

LOEWE, S., M.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City 1. *Research Prof., Emer.* (3, 1936)

LOEWI, OTTO, M.D. 155 E. 93rd St., New York City 28. *Research Prof. of Pharmacol., New York U. Coll. of Med.* (3H, 1941)

LOEWUS, FRANK A., Ph.D. USDA, Western Regional Research Lab., Albany 10, Calif. (2, 1956)

LOFTFIELD, ROBERT B., Ph.D. Massachusetts Gen. Hosp., Boston 14. *Assoc. Biochemist, Harvard U.* (2, 1957)

LOGAN, MILAN A., Ph.D. U. of Cincinnati Coll. of Med., Cincinnati 19, Ohio. *Prof. of Biol. Chem. and Head of Dept.* (2, 1936)

LO GRIPPO, GERALD A., M.D. Henry Ford Hosp., Div. of Microbiol., 2799 W. Grand Blvd., Detroit 2, Mich. *Sr. Chief.* (4, 1956; 6, 1953)

LOMBARD, LOUISE S., Ph.D., D.V.M. Argonne Natl. Lab., Biol. and Med. Div., Lemont, Ill. *Assoc. Pathologist.* (4, 1956)

LOMBARDO, MICHAEL E., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. in Biochem.* (2, 1957)

LONDON, IRVING M., M.D. Albert Einstein Coll. of Med., Dept. of Med., New York City 61. *Prof. and Chmn.* (2, 1958)

LONG, C. N. H., D.Sc., M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Sterling Prof. of Physiol.* (1, 1935; 2, 1927)

LONG, ESMOND R., M.D., Ph.D., Sc.D. Pedlar Mills, Va. *Prof. Emer. of Pathol., Henry Phipps Inst., U. of Pennsylvania.* (4R, 1930)

LONG, JOHN P., Ph.D. State U. of Iowa Coll. of Med., Dept. of Pharmacol., Iowa City. *Asst. Prof.* (3, 1956)

LONGENECKER, HERBERT E., Ph.D. U. of Illinois, Chicago 12, Ill. *Vice-Pres. in Charge of Chicago Professional Colleges.* (2, 1940; 5, 1945)

LONGWELL, BERNARD B., Ph.D. The Lovelace Fndn., 4800 Gibson Blvd., S. E., Albuquerque, N. Mex. *Head, Dept. of Biochem.* (2, 1946)

LOOFBOURROW, G. N., Ph.D. U. of Kansas, Dept. of Physiol., Lawrence. *Asst. Prof.* (1, 1950)

LOOMIS, TED A., Ph.D., M.D. U. of Washington, Sch. of Med., Dept. of Pharmacol., Seattle 5. (3, 1948)

LOOMIS, WILLIAM F., M.D. Calhoun Dr., Greenwich, Conn. *Dir., Loomis Lab.; Prof. of Biochem., Brandeis U.* (2, 1952)

LOONEY, JOSEPH M., M.D. 17 Court St., Boston, Mass. *Chief of Labs., VA Outpatient Clinic; Res. Prof. Biochem., Boston U.* (2, 1922)

LOOSLI, CLAYTON GARR, M.D., Ph.D. U. of Southern California, Los Angeles. *Dean, Sch. of Med.* (4R, 1940; 6, 1956)

LOOSLI, J. K., Ph.D. Cornell U., Animal Nutrition Lab., Ithaca, N. Y. *Prof. of Animal Nutrition.* (5, 1944)

LORAND, LASZLO, Ph.D. Northwestern U., Dept. of Chem., Evanston, Ill. *Assoc. Prof.* (1, 1955; 2, 1957)

LORBER, STANLEY H., M.D. Temple U. Sch. of Med., Philadelphia 40, Pa. *Asst. Prof. of Med.* (1, 1951)

LORBER, VICTOR, M.D. U. of Minnesota, Sch. of Med., Dept. of Physiol., Minneapolis 14. *Prof.; Career Invest., Amer. Heart Assn.* (1, 1944)

LORENTE DE NO, RAFAEL, M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (1, 1937)

LORING, H. S., Ph.D. Stanford U., Dept. of Chem., Stanford, Calif. *Prof. of Biochem.* (2, 1938)

LORING, WILLIAM E., M.D. New York U.-Bellevue Med. Ctr., Dept. of Pathol., New York City 16. *Assoc. Prof.* (4, 1957)

LOTSPEICH, WILLIAM D., M.D. U. of Cincinnati Coll. of Med., Dept. of Physiol., Cincinnati 19, Ohio. *Prof. and Chmn.* (1, 1948)

LOUCKES, HORTENSE S., Ph.D. U. of Tennessee, Dept. of Physiol., Memphis 3. *Asst. Prof.* (1, 1957)

LOUIS, LAWRENCE H., D.Sc. University Hosp., Dept. of Int. Med., Ann Arbor, Mich. *Assoc. Prof. of Biol. Chem., U. of Mich.* (2, 1949)

LOVE, ROBERT, M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Pathologist.* (4, 1958)

LOVE, WARNER E., Ph.D. Johns Hopkins U., Dept. of Biophysics, Baltimore 18, Md. *Asst. Prof.* (1, 1956)

LOVELESS, MARY H., M.D. New York Hosp., New York City 21. *Asst. Attend. Physician; Assoc. Prof. Clin. Med., Cornell U.* (6, 1941)

LOW, BARBARA W., D.Phil. Columbia U. Coll. of P. & S., New York City 32. *Assoc. Prof. of Biochem.* (2, 1958)

LOWE, CHARLES UPTON, M.D. U. of Buffalo Sch. of Med., Buffalo, N. Y. *Prof. of Pediatrics; Dir. of Res., Children's Hosp.* (4, 1954)

LOWELL, FRANCIS C., M.D. 65 E. Newton St., Boston, Mass. *Assoc. Prof. of Med., Boston U. Sch. of Med.* (6, 1942)

LOWENBACH, HANS, M.D. Duke U. Med. Sch., Dept. of Psychiatry, Durham, N. C. *Prof.* (1, 1946)

LOWRY, OLIVER H., M.D., Ph.D. Washington U. Sch. of Med., Dept. of Pharmacol., St. Louis 10, Mo. *Prof. and Head of Dept.* (2, 1942; 3, 1950)

LU, FRANK C., M.D. Dept. of Natl. Health and Welfare, Food and Drug Directorate, Ottawa, Canada. *Pharmacologist.* (3, 1951)

LU, GO, M.D., Ph.D. Ethicon, Inc., Dept. of Pharmacol., Somerville, N. J. *In Charge, Pharmacodynamics Sec.* (3, 1949)

LUBIN, MARTIN, M.D., Ph.D. Harvard Med. Sch., Dept. of Pharmacol., Boston 15, Mass. *Asst. Prof* (3, 1957)

LUBINSKI, HERBERT, M.D. Jewish Gen. Hosp., Montreal, Canada. *Bacteriologist and Serologist.* (6, 1941)

LUCAS, COLIN C., Ph.D. U. of Toronto, Banting and Best Dept. of Med. Research, Toronto, Canada. *Prof.* (2, 1946)

LUCAS, FRED V., M.D. Columbia U., Coll. of P. & S., Dept. of Pathol., New York City 32. *Assoc. Prof.* (4, 1955)

LUCAS, GEORGE H. W., Ph.D. U. of Toronto, Toronto, Canada. *Prof. of Pharmacol.* (2, 1925; 3, 1928)

LUCK, JAMES MURRAY, Ph.D. Stanford U., Dept. of Chem., Stanford, Calif. *Prof. of Biochem.* (2, 1925)

LUCKEY, THOMAS D., Ph.D. U. of Missouri Sch. of Med., Dept. of Biochem., Columbia. *Chmn.* (5, 1953)

LUDEWIG, STEPHAN, Ph.D. U. of Virginia Sch. of Med., Charlottesville. *Assoc. Prof. of Biochem.* (2, 1941)

LUDUENA, FROILAN P., Ph.D., M.D. Sterling-Winthrop Research Inst., Rensselaer, N. Y. *Research Pharmacologist.* (3, 1941)

LUECKE, RICHARD W., Ph.D. Michigan State U., Dept. of Agric. Chem., East Lansing. *Prof. (Research).* (5, 1950)

LUFT, ULRICH C., M.D. Lovelace Fndn. and Clinic, Albuquerque, N. Mex. *Head, Dept. of Physiol.* (1, 1950)

LUISADA, ALDO A., M.D. Chicago Med. Sch., Chicago 12, Ill. *Assoc. Prof. of Med.; Dir., Div. of*

Cardiol. (1, 1956)

LUKENS, FRANCIS D. W., M.D. U. of Pennsylvania, 811 Maloney Clinic, Philadelphia 4. *Prof. of Med.* (1, 1938)

LUMB, GEORGE D., M.D. U. of Tennessee Inst. of Pathol., Memphis 3. *Prof.* (4, 1958)

LUMRY, RUFUS, Ph.D. U. of Minnesota Sch. of Chem., Minneapolis 14. *Prof., Physical Chem.* (2, 1956)

LUNDBERG, WALTER O., Ph.D. Hormel Inst., Austin, Minn. *Dir.; Prof. of Agric. Biochem., U. of Minnesota.* (2, 1949)

LUNDGREN, HAROLD P., Ph.D. USDA, Western Regional Research Lab., Albany 10, Calif. *Acting Head, Wool & Mohair Lab.* (2, 1942)

LUNDY, JOHN SILAS, M.D. Mayo Clinic, Rochester, Minn. *Sr. Consultant.* (3, 1935)

LURIA, S. E., M.D. U. of Illinois, Urbana. *Prof. of Microbiol.* (6, 1958)

LURIE, MAX B., M.D. Henry Phipps Inst., Philadelphia 4, Pa. *Prof. of Exptl. Pathol.* (4, 1934; 6, 1930)

LUSE, SARAH A., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Assoc. Prof. of Pathol.* (4, 1956)

LUSHBAUGH, CLARENCE C., M.D., Ph.D. Los Alamos Sci. Lab., Los Alamos, N. Mex. *Staff Member; Pathologist, Los Alamos Hosp.* (4, 1950)

LUTZ, BRENTON R., Ph.D. Boston U., 675 Commonwealth Ave., Boston, Mass. *Prof. Emer. of Biol.* (1R, 1925)

LUYET, BASILE J., Sc.D. (Biol. and Physics). 3837 Monona Dr., Madison 4, Wis. (1, 1936)

LYMAN, CARL M., Ph.D. Texas A & M Coll., Dept. of Biochem. and Nutrition, College Station. *Prof. and Head of Dept.* (2, 1940; 5, 1952)

LYMAN, CHARLES PEIRSON, Ph.D. Harvard U., Museum of Comp. Zool., Cambridge, Mass. *Research Assoc., Museum & Med. Sch.* (1, 1951)

LYMAN, JOHN F., Ph.D. Red Bud Lane, RR. 13, Box 231, Indianapolis, Ind. *Emer. Prof. Agric. Biochem., Ohio State U.* (2R, 1920; 5R, 1933)

MAAS, WERNER K., Ph.D. New York U. Coll. of Med., Dept. of Microbiol., New York City 16. *Assoc. Prof.* (2, 1955)

MAASKE, CLARENCE A., Ph.D. U. of Colorado Sch. of Med., Dept. of Physiol., Denver 20. *Prof. and Head of Dept.* (1, 1945)

MACALLUM, A. BRUCE, M.D., Ph.D. R.R. 3, Lunenburg, Nova Scotia, Canada. (2R, 1914)

MacCANON, D. M., Ph.D. State U. of South Dakota Sch. of Med., Vermillion. *Asst. Prof. of Physiol. & Pharmacol.* (1, 1957)

MacCARDLE, ROSS C., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Prin. Cytologist.* (4, 1948)

MACCHI, I. ALDEN, Ph.D. Boston U., Boston 15, Mass. *Asst. Prof. of Biol.* (1, 1957)

MacCORQUODALE, D. W., Ph.D. Abbott Labs., North Chicago, Ill. *Head of Biochem. Research.* (2, 1934)

MacDONALD, DONALD LAURIE, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Visiting Scientist.* (2, 1956)

MacDONALD, NORMAN S., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Biophysics (in Residence).* (2, 1955)

MacFADYEN, DOUGLAS A., M.D. Presbyterian-St. Luke's Hosp., Dept. of Biochem., Chicago 12, Ill. *Chmn. of Dept.* (2, 1942)

MACHLIN, LAWRENCE J., Ph.D. Monsanto Chem. Co., Organic Chemicals Div., St. Louis 4, Mo. *Research Biochemist.* (5, 1958)

MACHT, DAVID ISRAEL, M.D. 6110 Biltmore Ave., Baltimore 15, Md. *Consultant Pharmacologist.* (1R, 1916; 3R, 1915)

MACHT, MARTIN B., Ph.D., M.D. U. of Cincinnati Coll. of Med., Cincinnati, Ohio. *Asst. Clin. Prof. of Med.* (1, 1948)

MacINTOSH, F. C., Ph.D. McGill U., Dept. of Physiol., Montreal, Canada. *Prof. and Chmn.* (1, 1950; 3, 1950)

MACK, WALTER N., Ph.D. Michigan State U., East Lansing. *Prof. of Microbiol. and Public Health.* (6, 1957)

MacKAY, EATON M., M.D. 110 N. China Lake Blvd., Ridgecrest, Calif. (1, 1930)

MACKAY, IAN F. S., Ph.D. Univ. Coll. of the West Indies, Jamaica, B. W. I. *Prof. of Physiol.* (1, 1949)

MACKENZIE, COSMO G., Sc.D. U. of Colorado Sch. of Med., Dept. of Biochem., Denver 20. *Prof. and Head.* (2, 1946; 5, 1942)

MACKENZIE, JULIA B., Sc.D. U. of Colorado Sch. of Med., Denver 20. *Assoc. Prof. of Biochem.* (2, 1952)

MACKINNEY, G., Ph.D. U. of California, 313 Hilgard Hall, Berkeley 4. *Prof. of Food Tech.* (2, 1958)

MACKLER, BRUCE, M.D. U. of Washington, Dept. of Pediatrics, Seattle 5. *Assoc. Prof.; Estab. Invest., Amer. Heart Assn.* (2, 1955)

MacLEOD, COLIN M., M.D. U. of Pennsylvania Hosp., John Herr Musser Dept. of Res. Med., Philadelphia 4. *Prof.* (6, 1937)

MacLEOD, FLORENCE L., Ph.D. U. of Tennessee, Knoxville. *Prof. of Nutrition.* (2, 1927; 5, 1933)

MacLEOD, GRACE, Ph.D. 860-20th St., Apt. 425S, Knoxville 16, Tenn. *Prof. Emer. of Nutrition, T. C., Columbia U.* (2R, 1924; 5R, 1933)

MacLEOD, JOHN, Ph.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Anatomy.* (1, 1942)

MacLEOD, ROBERT A., Ph.D. Fisheries Research Board of Canada, Tech. Sta., Vancouver 2, Canada. *Sr. Biochemist.* (2, 1953)

MACMILLAN, WILLIAM H., Ph.D. U. of Vermont Coll. of Med., Burlington. *Asst. Prof. of Pharmacol.* (3, 1957)

MacVICAR, ROBERT, Ph.D. Oklahoma State U., Stillwater. *Prof., Biol. Chem.; Dean, Grad. Sch. and Vice-Pres. for Acad. Affairs.* (2, 1949; 5, 1954)

MADDEN, SIDNEY C., M.D. U. of California Sch. of Med., Dept. of Pathol., Los Angeles 24. *Prof. and Chmn.* (4, 1939)

MADDOCK, CHARLOTTE L., M.D. Children's Med. Ctr. and Cancer Research Fndn., Boston 15, Mass. *Research Assoc. in Pathol.* (4, 1958)

MADDOCK, STEPHEN, M.D. Boston City Hosp., Boston, Mass. *Dir. of Surgical Research Lab.* (4, 1931)

MADDOCK, WILLIAM O., M.D., Ph.D. Wayne State U. Coll. of Med., Dept. of Med., Detroit 7, Mich. *Assoc. Prof.* (1, 1951)

MADSEN, LOUIS L., Ph.D. State Coll. of Washington, Inst. of Agric. Sci., Pullman. *Dir.* (5, 1940)

MAENGWYN-DAVIES, GERTRUDE D., Ph.D. 2909-34th St., N.W., Washington 8, D.C. *Assoc. Prof. Pharmacol., Georgetown U.* (2, 1957)

MAGASANIK, BORIS, Ph.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. Prof. of Bacteriol. and Immunol.* (2, 1952)

MAGATH, THOMAS B., Ph.D., M.D. Mayo Clinic, Rochester, Minn. *Sr. Consultant, Clin. Patbol.; Prof. Clin. Patbol & Parasitol., Mayo Fndn.* (1, 1928)

MAGEE, DONALD F., B.M., BcH., Ph.D. U. of Washington, Dept. of Pharmacol., Seattle 5. *Assoc. Prof.* (1, 1954; 3, 1957)

MAGILL, THOMAS P., M.D. State U. of New York Coll. of Med., Brooklyn 3. *Prof. of Microbiol. and Immunol.* (6, 1937)

MAGLADERY, J. W., M.D., Ph.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. of Neurol., Johns Hopkins U.* (1, 1950)

MAGNUSON, HAROLD J., M.D., M.P.H. U.S. Public Health Service, Washington 25, D.C. *Chief, Occupational Health Program.* (4, 1950)

MAGOUN, HORACE W., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Prof. of Anatomy.* (1, 1937)

MAHLER, HENRY R., Ph.D. Indiana U., Dept. of Chem., Bloomington. *Prof.* (2, 1953)

MAIN, ROLLAND J., Ph.D. Eaton Labs., Norwich, N.Y. *Dir., Div. Sci. Information and Consultant in Physiol.* (1, 1936)

MAIRE, FREDERICK WIRTH, M.D. Mental Health Research Inst., Fort Steilacoom, Wash. *Physician II in Clin. Neurophysiol.* (1, 1957)

MAISON, GEORGE L., M.D. Riker Labs., Northridge, Calif. *Pres.* (1, 1939; 3, 1948)

MAJNO, GUIDO, M.D. Harvard Med. Sch., Boston 15, Mass. *Asst. in Patbol.* (4, 1956)

MAJOR, RANDOLPH T., Ph.D. U. of Virginia, Charlottesville. *Prof. of Chem.; Sci. Advisor, Merck & Co., Inc.* (2, 1942)

MAKINODAN, TAKASHI, Ph.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Biologist.* (6, 1956)

MALEY, GLADYS FELDOTT, Ph.D. New York State Dept. of Health, Div. of Labs. and Research, Albany 1. *Research Fellow.* (2, 1957)

MALING, HARRIET M., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Pharmacologist.* (3, 1953)

MALIS, LEONARD I., M.D. 1158 Fifth Ave., New York City. *Assoc. Attending Neurosurgeon, Mt. Sinai Hosp.* (1, 1958)

MALKIEL, SAUL, M.D., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Res. Assoc. in Med.; Assoc. in Med., Peter Bent Brigham Hosp.* (6, 1948)

MALLETTE, M. F., Ph.D. Pennsylvania State U., University Park. *Assoc. Prof. of Physical Biochem.* (2, 1954)

MALLORY, G. KENNETH, M.D. Boston City Hosp., Mallory Inst. of Pathol., Boston, Mass. *Pathologist-in-Chief.* (4, 1940)

MALLOV, SAMUEL, Ph.D. State U. of New York Coll. of Med., Upstate Med. Ctr., Dept. of Pharmacol., Syracuse 10. *Asst. Prof.* (3, 1956)

MALMGREN, RICHARD A., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. (4, 1955)

MALONEY, JAMES V., JR., M.D. U. of California Sch. of Med., Dept. of Surgery, Los Angeles 24. *Asst. Prof.* (1, 1951)

MALTANER, FRANK, Ph.D. 388 New Scotland Ave., Albany, N.Y. (6R, 1920)

MALUF, NOBLE S. R., Ph.D., M.D. 4867 Sunset Blvd., Los Angeles 27, Calif. (1, 1942)

MAN, EVELYN B., Ph.D. Yale U. Sch. of Med., Dept. of Med., New Haven, Conn. *Asst. Prof. in Thyroid Lab.* (2, 1936)

MANDEL, H. GEORGE, Ph.D. George Washington U. Sch. of Med., Dept. of Pharmacol., Washington 5, D.C. *Prof.* (2, 1953; 3, 1955)

MANDL, INES, Ph.D. 166 W. 72nd St., New York City 23. *Asst. Prof. of Biochem., Columbia U., Coll. of P. & S.* (2, 1952)

MANERY, JEANNE FOREST, Ph.D. U. of Toronto Med. Sch., Toronto, Canada. *Asst. Prof. of Biochem.* (1, 1937)

MANGUN, GEORGE H., Ph.D. Schoolhouse Lane, Morristown, N.J. *Biochem. and Drug Research; Consulting.* (2, 1947)

MANIRE, GEORGE PHILIP, Ph.D. U. of North Carolina Sch. of Med., Dept. of Bacteriol., Chapel Hill. *Assoc. Prof.* (6, 1953)

MANN, FRANK C., M.D. 605-11th St., S.W., Rochester, Minn. *Prof. Emer. Exptl. Med., Mayo Fndn.* (1R, 1916; 4R, 1924)

MANN, FRANK D., Ph.D., M.D. St. Joseph's Hosp., Phoenix, Ariz. (1, 1950; 4, 1950)

MANN, GEORGE V., Sc.D., M.D. Vanderbilt U., Nashville 5, Tenn. *Assoc. Prof., Biol.; Asst. Prof., Med.* (5, 1951)

MANN, JOSEPH DANIEL, M.D. Butterworth Hosp., Grand Rapids 3, Mich. *Dir. of Labs.* (4, 1953)

MANNERING, GILBERT J., Ph.D. U. of Wisconsin, Dept. of Pharmacol. and Toxicol., Madison 6. *Asst. Prof.* (3, 1956)

MANNING, G. W., M.D., Ph.D. Victoria Hosp., London, Canada. *Asst. Prof., Clin. Invest.; Lect., Dept. of Physiol., U. of Western Ontario.* (1, 1944)

MANSOUR, TAG E., Ph.D. Louisiana State U. Sch. of Med., New Orleans 12. *Asst. Prof. of Pharmacol.* (3, 1956)

MANTHEI, R. W., Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Assoc. Prof. of Pharmacol.* (3, 1957)

MANVILLE, IRA ALBERT, M.D., Ph.D. 811 N.W. 19th Ave., Portland 9, Oreg. (1, 1933)

MANWARING, WILFRED H., M.D. 364 Kingsley Ave., Palo Alto, Calif. *Prof. Emer. Bacteriol. and Exptl. Patbol., Stanford U.* (4, prior to 1920; 6, 1917)

MARBARGER, JOHN P., Ph.D. 394 S. Kenilworth Ave., Elmhurst, Ill. *Res. Dir., Aeromed. & Phys. Environ. Lab.; Prof. of Physiol.* (1, 1949)

MARCUS, STANLEY, Ph.D. U. of Utah, Dept. of Bacteriol., Salt Lake City. *Assoc. Prof.* (6, 1948)

MAREN, THOMAS H., M.D. U. of Florida Coll. of Med., Dept. of Pharmacol., Gainesville. *Prof. and Chmn.* (3, 1950)

MARGOLIN, SOLOMON, Ph.D. Wallace Labs., Dept. of Pharmacol., New Brunswick, N.J. *Chief Pharmacologist.* (3, 1957)

MARGOLIS, GEORGE, M.D. Duke U. Sch. of Med., Durham, N.C. *Prof. of Patbol.* (4, 1957)

MARINE, DAVID, M.D. 18 Baltimore Ave., Rehoboth, Del. (1R, 1910; 4R, 1913)

MARINETTI, GUIDO V., Ph.D. U. of Rochester Sch.

of Med., Dept. of Biochem., Rochester 20, N.Y. *Asst. Prof.* (2, 1956)

MARK, LESTER C., M.D. Columbia U., Coll. of P. & S., Dept. of Anesthesiol., New York City 32. *Assoc. Prof.* (3, 1950)

MARKEE, JOSEPH E., Ph.D. Duke U. Sch. of Med., Durham, N.C. *Prof. of Anatomy.* (1, 1945)

MARKOWITZ, J., M.D., Ph.D., M.S.(Exptl. Surg.) U. of Toronto Sch. of Med., Toronto, Canada. *Prof. of Physiol.* (1, 1929)

MARKS, PAUL A., M.D. Columbia U., Coll. of P. & S., New York City 32. *Asst. Prof. of Med.* (2, 1957)

MARMONT, GEORGE H., Ph.D. Box 788, Monterey, Calif. (1, 1941)

MARMORSTON, JESSIE. 613 N. Canon Dr., Beverly Hills, Calif. (6, 1932)

MARMUR, JULIUS, Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Research Fellow.* (2, 1958)

MARAZZI, AMEDEO S., M.D. VA Res. Labs. in Neuropsychiatry, Pittsburgh 6, Pa. *Dir.; Prof. Physiol. & Pharmacol., U. Pittsburgh.* (1, 1949; 3, 1938)

MARSH, GORDON, Ph.D. State U. of Iowa, Iowa City. *Prof. of Zool.* (1, 1944)

MARSH, JULIAN B., M.D. U. of Pennsylvania Sch. of Med. and Grad. Sch. of Med., Dept. of Biochem., Philadelphia 4. *Asst. Prof.* (2, 1955)

MARSH, M. ELIZABETH, Ph.D. U. of California Med. Ctr., Dept. of Pathol., San Francisco 22. *Asst. Res. Pathologist.* (1, 1929; 5, 1933)

MARSHAK, ALFRED GEORGE, Ph.D. Marine Biological Lab., Woods Hole, Mass. (1, 1940)

MARSHALL, ELI K., JR., Ph.D., M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Prof. Emer.* (1R, 1915; 2R, 1913; 3, 1915)

MARSHALL, JEAN McELROY, Ph.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Physiol.* (1, 1956)

MARSHALL, LAWRENCE M., Ph.D. Howard U., Sch. of Med., Dept. of Biochem., Washington 1, D.C. *Assoc. Prof.* (2, 1952)

MARSHALL, LOUISE HANSON, Ph.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Physiologist.* (1, 1946)

MARSHALL, WADE H., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Neurophysiologist.* (1, 1937)

MARTIN, A. ROBERT, Ph.D. U. of Utah Coll. of Med., Dept. of Physiol., Salt Lake City. *Instr.* (1, 1957)

MARTIN, ARTHUR W., Ph.D. U. of Washington, Dept. of Zool., Johnson Hall, Seattle 1. *Exec. Officer.* (1, 1944)

MARTIN, DONALD S., M.D. Communicable Disease Ctr., 50-7th St., N.E., Atlanta 23, Ga. *Chief, Training Br.* (6, 1943)

MARTIN, FOSTER N., JR., Ph.D., M.D. 1546 Marlin St., Newberry, S.C. *Practice of Med.* (3, 1947)

MARTIN, GUSTAV J., Sc.D. Natl. Drug Co., Research Labs., Haines & MacCallum Sts., Philadelphia, Pa. *Res. Dir.* (2, 1951)

MARTIN, SAMUEL P., M.D. U. of Florida Coll. of Med., Gainesville. *Prof. of Med.* (6, 1954)

MARTIN, STEVENS J., Ph.D., M.D. St. Francis Hosp., Hartford, Conn. *Dir., Dept. of Anesthesiol.* (1, 1933)

MARTINEZ, CARLOS, M.D., Ph.D. U. of Minnesota, Dept. of Physiol., Minneapolis 14. *Assoc. Prof. in Cancer Biol.* (1, 1955)

MARX, WALTER, Ph.D. U. of Southern California Sch. of Med., Los Angeles 7. *Prof. of Biochem.* (2, 1949)

MASON, EARL JAMES, Ph.D., M.D. VA Hosp., Pathol. Dept., Temple, Tex. (4, 1956)

MASON, ELEANOR DEWEY, Ph.D., D.Sc. U. of Bombay, Dept. of Chem. Technol., Bombay 19, India. *Research Assoc.* (1, 1946)

MASON, HAROLD L., Ph.D. Mayo Clinic, Rochester, Minn. *Prof. of Physiol. Chem., Mayo Fndn., U. of Minnesota.* (2, 1941)

MASON, HERMAN C., Ph.D. 714 Hamlin St., Seattle 2, Wash. *Dir., Public Health Labs., State Dept. of Health.* (6, 1948)

MASON, HOWARD S., Ph.D. U. of Oregon Med. Sch., Portland 1. *Assoc. Prof. of Biochem.* (2, 1949)

MASON, KARL ERNEST, Ph.D. U. of Rochester Sch. of Med. and Dent., Dept. of Anatomy, Rochester 20, N.Y. *Prof. and Chmn.* (5, 1941)

MASON, MERLE, Ph.D. U. of Michigan, Dept. of Biol. Chem., Ann Arbor. *Asst. Prof.* (2, 1958)

MASON, MORTON F., Ph.D. Parkland Mem. Hosp., Dallas 35, Tex. *Prof., Clin. Chem., U. of Texas Southwestern Med. Sch.* (2, 1938)

MASON, RICHARD P., M.D. Walter Reed Army Inst. of Research, Washington 12, D.C. (6, 1950)

MASORO, EDWARD J., Ph.D. Tufts U. Sch. of Med., Dept. of Physiol., Boston 11, Mass. *Assoc. Prof.* (1, 1954)

MASOUREDIS, SERAFEIM P., M.D., Ph.D. U. of Pittsburgh, Pittsburgh 13, Pa. *Assoc. Prof.; Asst. Dir., Central Blood Bank.* (1, 1957; 6, 1957)

MASSEY, VINCENT, Ph.D. U. of Sheffield, Biochem. Dept., Sheffield, England. *Lect.* (2, 1956)

MASSON, GEORGES M. C., Ph.D. Cleveland Clinic Fndn., Research Div., Cleveland 6, Ohio. *Staff Member.* (1, 1944)

MATHEWS, MARTIN B., Ph.D. U. of Chicago, Dept. of Pediatrics, Chicago 37, Ill. *Res. Assoc.* (2, 1954)

MATHIES, JAMES C., Ph.D. Swedish Hosp., Summit and Columbia, Seattle, Wash. (2, 1952)

MATHIESON, DON R., M.D. U. of Minnesota, Mayo Clinic, Rochester. *Asst. Prof. and Consultant in Clin. Pathol.* (6, 1952)

MATRONE, GENNARD, Ph.D. North Carolina State Coll., Animal Nutrition Sec., Raleigh. *Research Prof. of Animal Industry.* (5, 1957)

MATSON, G. ALBIN, Ph.D. Minneapolis War Memorial Blood Bank, Inc., 2304 Park Ave., Minneapolis 4, Minn. *Dir.* (6, 1946)

MATTERSON, LLOYD D., Ph.D. U. of Connecticut, Dept. of Poultry Sci., Storrs. *Prof.* (5, 1955)

MATTHEWS, SAMUEL A., Ph.D. Williams Coll., Williamstown, Mass. *Prof. of Biol.* (1, 1948)

MATTIS, PAUL A., D.Sc. Smith, Kline & French Labs., Philadelphia 1, Pa. *Head of Pharmacol. Sec.* (3, 1946)

MATTOX, VERNON ROSS, Ph.D. Mayo Clinic, Dept. of Biochem., Rochester, Minn. *Assoc. Prof.* (2, 1954)

MATTSON, FRED H., Ph.D. Procter & Gamble Co., Research Div., Cincinnati 2, Ohio. *Research Chemist.* (2, 1957; 5, 1953)

MAURER, FRANK W., Ph.D. 301 Lake Ave., Newton Highlands 61, Mass. (1, 1941)

MAURER, FRED D., D.V.M., Ph.D. Armed Forces Inst. of Pathol., Washington 12, D.C. (6, 1950)

MAURER, PAUL H., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Pathol., Pittsburgh 13, Pa. *Assoc. Prof. of Immunochem.* (6, 1953)

MAUTZ, FREDERICK R., M.D. 10515 Carnegie Ave., Cleveland 6, Ohio. (1, 1945)

MAVER, MARY E., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1947)

MAVOR, JAMES WATT, Ph.D. 8 Gracewood Pk., Cambridge, Mass. (1R, 1930)

MAXFIELD, MARY E., Ph.D. Haskell Labs., E.I. duPont de Nemours & Co., Newark, Del. (1, 1947)

MAXFIELD, MYLES, M.D., Ph.D. Brookhaven Natl. Lab., Med. Dept., Upton, N.Y. *Assoc. Scientist.* (1, 1955)

MAXWELL, ELIZABETH S., Sc.D. Natl. Insts. of Health, Bethesda 14, Md. *Chemist.* (2, 1958)

MAXWELL, MORTON H., M.D. U. of California Med. Ctr., Los Angeles. *Assoc. Clin. Prof. of Med.* (1, 1955)

MAXWELL, ROBERT A., Ph.D. Ciba Pharmaceutical Products, Inc., Macrobiol. Dept., Summit, N.J. *Assoc. Pharmacologist.* (3, 1957)

MAY, CHARLES D., M.D. 667 Madison Ave., New York City 21. *Editor, "Pediatrics".* (5, 1952)

MAYER, DENNIS T., Ph.D. U. of Missouri, Dept. of Agric. Chem., 211 N. Schweitzer Hall, Columbia. *Prof.* (2, 1950)

MAYER, JEAN, Sc.D., Ph.D. Harvard Sch. of Public Health, Dept. of Nutrition, Boston 15, Mass. *Assoc. Prof.* (1, 1955; 5, 1951)

MAYER, MANFRED M., Ph.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Microbiol.* (6, 1946)

MAYER, STEVEN E., Ph.D. Emory U., Div. Basic Health Sci., Dept. of Pharmacol., Atlanta 22, Ga. *Asst. Prof.* (3, 1957)

MAYERSON, HYMEN S., Ph.D. Tulane U. Sch. of Med., Dept. of Physiol., University Sta., New Orleans 18, La. *Prof. and Head.* (1, 1928)

MAYNARD, ELLIOTT ALLEN, Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Assoc. Prof. of Pharmacol. and Toxicol.* (3, 1950)

MAYNARD, L. A., Ph.D. Cornell U., Ithaca, N.Y. *Prof. Emer. of Nutrition and Biochem.* (2R, 1930; 5, 1933)

MAYNERT, EVERETT W., Ph.D., M.D. Johns Hopkins U. Sch. of Med., Dept. of Pharmacol., Baltimore 5, Md. *Assoc. Prof.* (3, 1952)

MAZUR, ABRAHAM, Ph.D. City Coll. of New York, Dept. of Chem., New York City 31. *Assoc. Prof.* (2, 1944)

McCANDLESS, ESTHER LEIB, Ph.D. U. of Tennessee, Dept. of Physiol., Memphis 3. *Asst. Prof.* (1, 1957)

McCANN, SAMUEL McD., M.D. U. of Pennsylvania Sch. of Med., Dept. of Physiol., Philadelphia 4. *Assoc. Prof.* (1, 1955)

McCANN, WILLIAM PETER, M.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Pharmacol.* (3, 1958)

McCANN, WILLIAM S., M.D. U. of Rochester Sch. of Med., Rochester 20, N.Y. *Charles A. Dewey Prof. of Med., Emer.* (2R, 1923; 5, 1933)

McCARRELL, JANE D., Ph.D. Hood Coll., Dept. of Biol., Frederick, Md. *Prof. and Chmn. of Dept.* (1, 1942)

McCARTHY, MILES D., Ph.D. Pomona Coll., Dept. of Zool., Claremont, Calif. *Prof.* (1, 1955)

McCARTY, MACLYN, M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (6, 1947)

McCAWLEY, ELTON LEEMAN, Ph.D. U. of Oregon Sch. of Med., Portland 1. *Assoc. Prof. of Pharmacol.* (3, 1944)

McCAY, CLIVE M., Ph.D. Cornell U., Animal Nutrition Lab., Dairy Bldg., Ithaca, N.Y. *Prof. of Nutrition.* (2, 1929; 5, 1933)

McCHESNEY, EVAN WILLIAM, Ph.D. Sterling-Winthrop Research Inst., Rensselaer, N.Y. *Member, Biol. Div.* (1, 1944)

McCLAIN, MARY ELIZABETH, Ph.D. U. of California, Naval Biol. Lab., Berkeley. *Assoc. Research Virologist.* (6, 1957)

McCLELLAN, WALTER S., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Lect. in Physiol.* (1, 1931)

McCLENDON, J. F., Ph.D. Route 1, Box 393, Norristown, Pa. *Biochemist; X-Ray, Albert Einstein Med. Ctr., N.D.* (1R, 1910; 2R, 1914; 5R, 1935)

McCLUNG, L. S., Ph.D. Indiana U., Dept. of Bacteriol., Bloomington. *Prof. and Chmn.* (6, 1948)

McCLURE, FRANK J., Ph.D. Natl. Inst. of Dental Res., Lab. of Oral & Biol. Chem., NIH, Bethesda 14, Md. *Chief.* (5, 1958)

McCOLL, JOHN D., Ph.D. Frank W. Horner, Ltd., Box 959, Montreal, Canada. *Pharmacologist.* (3, 1957)

McCOLLUM, ELMER VERNER, Ph.D. Johns Hopkins U., Baltimore 18, Md. *Prof. Emer. of Biochem.* (2R, 1910; 5, 1933)

McCOLLUM, ERNESTINE BECKER, M.A. Johns Hopkins U. Sch. of Hygiene, Baltimore 5, Md. *Asst. Prof. of Biochem.* (5, 1938)

McCONNELL, KENNETH P., Ph.D. VA Hosp., Louisville, Ky. *Prin. Scientist, Radioisotope Serv.* (2, 1952)

McCOUCH, GRAYSON PREVOST, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Emer. Prof. of Physiol.* (1, 1925)

McCOY, RICHARD H., Ph.D. U. of Pittsburgh, Dept. of Chem., Pittsburgh 13, Pa. *Assoc. Prof.* (2, 1950; 5, 1948)

McCREA, FORREST D., Ph.D. 1023 Demerius St., Durham, N.C. (3, 1937)

McCRUDDEN, FRANCIS H., M.D. 19 Stoneleigh Rd., West Newton 65, Mass. (2R, 1906)

McCUBBIN, JAMES W., M.D. Cleveland Clinic Fndn., 2020 E. 93rd St., Cleveland 6, Ohio. *Staff, Research Div.* (1, 1956)

McCULLOCH, WARREN STURGIS, M.D. Massachusetts Inst. of Tech., Research Lab. of Electronics, Cambridge 39. *Staff Member.* (1, 1936)

McCUTCHEON, F. HAROLD, Ph.D. U. of Pennsylvania, Philadelphia 4. *Prof. of Physiol.* (1, 1951)

McCUTCHEON, MORTON, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Emer. Prof. of Patbol.* (4, 1925)

McDONALD, FRANCIS GUY, Ph.D. Mead Johnson &

Co., Research Labs., Evansville, Ind. *Dir., Control Lab.* (2, 1936; 5, 1947)

McDONALD, HUGH J., D.Sc. Loyola U., Stritch Sch. of Med., Dept. of Biochem., Chicago 12, Ill. *Prof. and Chmn. of Dept.* (2, 1954)

McDONALD, ROGER K., M.D. 10112 Parkwood Terr., Bethesda 14, Md. (1, 1949)

McELLROY, WILLIAM SWINDLER, M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Dean; Prof. of Biochem.* (2, 1919)

McELROY, L. W., Ph.D. U. of Alberta, Dept. of Animal Sci., Edmonton, Canada. *Prof.* (5, 1944)

McELROY, WILLIAM D., Ph.D. Johns Hopkins U., McCollum-Pratt Inst., Dept. of Biol., Baltimore 18, Md. *Dir. and Head of Dept.* (2, 1951)

McFARLAND, ROSS A., Ph.D. Harvard U. Sch. of Public Health, Boston 15, Mass. *Prof., Environ. Health and Safety.* (1, 1943)

McFARLANE, WILLIAM DOUGLAS, Ph.D. Canadian Breweries, Ltd., 307 Fleet St. E., Toronto, Canada. *Dir. of Research.* (2, 1933)

McGANITY, WILLIAM J., M.D. Vanderbilt U. Sch. of Med., Nashville, Tenn. *Assoc. Prof. of Obstetrics and Gynecol.* (5, 1957)

McGILL, HENRY C., JR., M.D. Louisiana State U. Sch. of Med., New Orleans 12. *Assoc. Prof. of Pathol.* (4, 1955)

McGILVERY, ROBERT W., Ph.D. U. of Virginia Sch. of Med., Dept. of Biochem., Charlottesville. *Assoc. Prof.* (2, 1951)

McGINNIS, JAMES, Ph.D. State Coll. of Washington, Dept. of Poultry Sci., Pullman. *Prof. of Nutrition and Chmn. of Dept.* (5, 1952)

McGINTY, DANIEL A., Ph.D. Parke, Davis & Co., Detroit, Mich. *Lab. Dir.* (1, 1925)

McGUIGAN, HUGH ALISTER, Ph.D., M.D. 2418 Park Pl., Evanston, Ill. *Prof. Emer., Pharmacol., U. of Illinois.* (1R, 1907; 2R, 1906; 3R, 1913)

McHARGUE, J. S., Ph.D. U. of Kentucky, Dept. of Chem., Kentucky Agric. Expt. Sta., Lexington. *Member Emer.* (2R, 1927)

McHENRY, E. W., Ph.D. U. of Toronto, Sch. of Hygiene, Toronto, Canada. *Prof. of Public Health Nutrition.* (2, 1938; 5, 1935)

McINTIRE, FLOYD C., Ph.D. Abbott Labs., North Chicago, Ill. *Asst. Head of Biochem.* (2, 1952)

McINTYRE, A. R., Ph.D., M.D. U. of Nebraska Coll. of Med., Dept. of Physiol. & Pharmacol., Omaha 5. *Prof. and Chmn. of Dept.* (1, 1933; 3, 1938)

McKAY, DONALD GEORGE, M.D. Harvard Med. Sch., Dept. of Pathol., Boston 15, Mass. *Pathologist, Free Hosp. for Women, Brookline.* (4, 1956)

McKEE, ALBERT P., M.D. State U. of Iowa Coll. of Med., Iowa City. *Prof. of Bacteriol.* (6, 1948)

McKEE, CLARA M. 6813 Blenheim Rd., Baltimore 12, Md. (6, 1941)

McKEE, FRANK W., M.D. U. of California Sch. of Med., Dept. of Pathol., Los Angeles 24. *Prof.* (4, 1947)

McKEE, RALPH WENDELL, Ph.D. U. of California Med. Ctr., Dept. of Physiol. Chem., Los Angeles 24. *Prof.* (2, 1946)

McKENNIS, HERBERT, JR., Ph.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Pharmacol.* (2, 1948; 3, 1957)

McKIBBIN, JOHN M., Ph.D. State U. of New York, Upstate Med. Ctr., Syracuse. *Prof. of Biochem.* (2, 1948)

McKINNEY, GORDON R., Ph.D. West Virginia U. Med. Ctr., Dept. of Pharmacol., Morgantown. *Assoc. Prof.* (3, 2954)

McLAIN, PAUL L., M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Prof. of Physiol. and Pharmacol.* (1, 1948; 3, 1940)

McLAREN, A. DOUGLAS, Ph.D. U. of California Coll. of Agric., 108 Hilgard Hall, Berkeley 4. *Prof. of Soil Chem.* (2, 1953)

McLAREN, BARBARA A., Ph.D. U. of Toronto, Dept. of Household Sci., Toronto, Canada. *Prof. and Dir. of Fac.—Household Sci.* (5, 1954)

McLEAN, FRANKLIN C., Ph.D., M.D. U. of Chicago, Chicago 37, Ill. *Prof. Emer. of Physiol.* (1, 1914; 2R, 1916; 3R, 1916)

McLEAN, I. WILLIAM, JR., M.D. Parke, Davis & Co., Virus Research Div., Detroit, Mich. *Asst. Dir., Microbiol. Research.* (6, 1946)

McLEAN, RICHARD A., M.A. Smith, Kline & French Labs., Philadelphia 1, Pa. *Sr. Pharmacologist.* (3, 1953)

McLIMANS, WILLIAM F., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Research Assoc. Prof.* (6, 1949)

McMANUS, IVY ROSABELLE, Ph.D. U. of Pittsburgh Grad. Sch. of Public Health, Dept. Biochem. and Nutrition, Pittsburgh 13, Pa. *Asst. Res. Prof.* (2, 1958)

McMANUS, J. F. A., M.D. U. of Alabama Med. Ctr., Dept. of Pathol., Birmingham. *Prof. and Chmn. of Dept.* (4, 1948)

McMASTER, PHILIP D., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (4, 1924; 6, 1951)

McMEEKIN, THOMAS L., Ph.D. USDA, Eastern Regional Research Lab., Philadelphia, Pa. *Head of Protein Div.* (2, 1935)

McNAMARA, BERNARD P., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Chief, Toxicol. Div.* (3, 1947)

McNAUGHT, JAMES BERNARD, M.D. U. of Colorado Sch. of Med., Dept. of Pathol., Denver 20. *Prof. and Head.* (4, 1936)

McNEIL, CRICHTON, M.D. Holy Cross Hosp., Salt Lake City, Utah. *Pathologist and Dir.* (6, 1950)

McNELLY, W. C., Ph.D. Miami U., Oxford, Ohio. *Prof. of Physiol.* (1, 1955)

McPHAIL, MURCHIE K., Ph.D. Natl. Defence Board, Suffield Expt. Sta., Ralston, Alberta, Canada. *Research Scientist.* (3, 1941)

McQUARRIE, IRVINE, Ph.D., M.D. Children's Hosp., Oakland 9, Calif. *Dir., Pediatric Res.; Prof. Emer. Pediatrics, U. Minn.* (1, 1949; 4, 1927; 5R, 1933)

McSHAN, W. H., Ph.D. U. of Wisconsin, Zool. Dept., Madison 6. *Prof. of Zoochem.* (2, 1947)

MEAD, JAMES F., Ph.D. 1210 Las Lomas Pl., Pacific Palisades, Calif. *Chief, Biochem. Div., Atomic Energy Proj. and Prof., UCLA.* (2, 1952)

MEAD, JERE, M.D. Harvard Sch. of Public Health, Physiol. Dept., Boston 15, Mass. *Assoc. Prof.* (1, 1952)

MEDES, GRACE, Ph.D. Lankenau Hosp. Research Inst., Philadelphia 11, Pa. *Sr. Member, Metabolic Chem.* (2, 1930)

MEEK, WALTER J., Ph.D., Sc.D. U. of Wisconsin,

Madison. *Emer. Prof. of Physiol.; Emer. Dean of Med. Sch.* (1R, 1908)

MEHL, JOHN W., Ph.D. U. of Southern California, Los Angeles. *Prof. of Biochem. and Nutrition.* (2, 1946)

MEHLER, ALAN H., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Chemist.* (2, 1952)

MEIER, ROLF, M.D. Bundestrasse 21, Basle, Switzerland. *Prof. of Pathol. Physiol.; Dir., Biol. Research Labs., Ciba Ltd.* (3, 1949)

MEIKLEJOHN, GORDON, M.D. U. of Colorado Med. Ctr., Dept. of Med., Denver 20. *Prof. and Head of Dept.* (6, 1948)

MEISSNER, WILLIAM A., M.D. New England Deaconess Hosp., Boston 15, Mass. *Pathologist; Assoc. Clin. Prof. Pathol., Harvard U.* (4, 1957)

MEISTER, ALTON, M.D. Tufts U. Sch. of Med., Dept. of Biochem., Boston, Mass. *Prof. and Chmn. of Dept.* (2, 1950)

MEITES, JOSEPH, Ph.D. Michigan State U., Dept. of Physiol. and Pharmacol., East Lansing. *Prof.* (1, 1949; 5, 1952)

MELAMPY, ROBERT M., Ph.D. Iowa State Coll., 33 Curtiss Hall, Ames. *Prof. of Physiol.* (1, 1952)

MELCHER, LEO R., M.D., Ph.D. 2900 Broadway, Redwood City, Calif. (6, 1952)

MELCHIOR, JACKLYN B., Ph.D. Loyola U., Stritch Sch. of Med., Chicago 12, Ill. *Assoc. Prof. of Pharmacol.* (2, 1953)

MELLETT, LAWRENCE B., Ph.D. U. of Michigan Med. Sch., Dept. of Pharmacol, Ann Arbor. *Instr.* (3, 1958)

MELLON, RALPH R., M.D., D.P.H. 6579 Mission Ave., 29 Palms, Calif. (6R, 1918)

MELLORS, ROBERT C., M.D., Ph.D. Memorial Ctr., New York City 21. *Assoc. Pathologist; Assoc., Sloan-Kettering Inst.* (2, 1951; 4, 1955; 6, 1957)

MELNICK, DANIEL, Ph.D. The Best Foods, Inc., Research Labs., Bayonne, N. J. *Chief Technologist.* (2, 1940; 5, 1942)

MELNICK, JOSEPH L., Ph.D. Baylor U., Coll. of Med., Virol. and Epidemiol. Dept., Houston 25, Tex. *Prof. & Chmn.* (6, 1948)

MELVILLE, DONALD B., Ph.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Biochem.* (2, 1947)

MELVILLE, KENNETH IVAN, M.D. McGill U., Dept. of Pharmacol., Montreal, Canada. *Prof. and Chmn. of Dept.* (3, 1931)

MENDEL, BRUNO, M.D. U. of Amsterdam, Pharmacotherap. Lab., Polderweg 20, Amsterdam, Holland. *Prof. and Head, Dept. Pharmacol.* (2, 1947)

MENDELSON, E. S., A.B. R.D. 2, Telford, Pa. *Physiologist, Air Crew Equip. Lab., Naval Air Material Ctr., Philadelphia.* (1, 1949)

MENDEZ, CARLOS, M.D. Instituto Nacional de Cardiologia, Ave. Cuauhtemoc 300, Mexico, D. F. 7. *Research Assoc.* (3, 1958)

MENDEZ, RAFAEL, M.D. Natl. Inst. of Cardiol., Avenida Cuauhtemoc 300, Mexico D.F., Mexico. *Head of Dept. of Pharmacol.* (3, 1944)

MENDLOWITZ, MILTON, M.D. 2 E. 95th St., New York City 28. *Assoc. Attending Physician, Mt. Sinai Hosp.* (1, 1949)

MENEELY, GEORGE R., M.D. Vanderbilt U. Hosp., Nashville 5, Tenn. *Dir., Clin. Radioisotope Ctr.; Assoc. Prof. Med.* (1, 1951; 4, 1946)

MENG, H. C., M.D., Ph.D. Vanderbilt U. Sch. of Med., Dept. of Physiol., Nashville 5. *Assoc. Prof.* (1, 1950)

MENKIN, V., M.D. Temple U. Sch. of Med., Philadelphia, Pa. *Assoc. Prof. Exptl. Pathol.; Head, Exptl. Pathol., Chase Fndn.* (1, 1932; 4, 1932; 6, 1931)

MENTEN, MAUD L., M.D., Ph.D. 8 Marlboro St., West, Leamington, Ont., Canada. *Ret. Prof. of Pathol., U. of Pittsburgh.* (1R, 1915; 4, 1927)

MERRICK, ARTHUR WEST, Ph.D. U. of Missouri Med. Ctr., Dept. of Physiol. and Pharmacol., Columbia. *Asst. Prof. of Physiol.* (1, 1958)

MERRIFIELD, ROBERT B., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (2, 1955)

MERRILL, JOHN P., M.D. Peter Bent Brigham Hosp., Boston 15, Mass. *Sr. Assoc. in Med.; Asst. Prof., Med., Harvard U.* (1, 1957)

MERTZ, EDWIN T., Ph.D. Purdue U., Dept. of Biochem., Lafayette, Ind. *Prof.* (2, 1950; 5, 1954)

METTIER, STACY R., M.D. U. of California Hosp., San Francisco. *Prof. of Med.* (4, 1932)

METTLER, FRED A., Ph.D., M.D., Sc.D. Columbia U., Coll. of P. & S., Dept. of Neurol., New York City 32. *Prof. of Anatomy.* (1, 1937)

METZLER, DAVID E., Ph.D. Iowa State Coll., Dept. of Chem., Ames. *Assoc. Prof.* (2, 1957)

MEYER, ANDRE S., Ph.D. Schering Corp., Bloomfield, N. J. *Sr. Research Biochemist.* (2, 1958)

MEYER, ARTHUR E., Ph.D. Hunt, Tex. (1R, 1948)

MEYER, CURTIS E., Ph.D. Upjohn Co., Kalamazoo, Mich. *Sr. Scientist in Nutrition and Metabolic Diseases.* (2, 1942)

MEYER, DALLAS K., Ph.D. 401 Crestland Rd., Columbia, Mo. *Prof. of Physiol., U. of Missouri Sch. of Med.* (1, 1951)

MEYER, FRIEDA L., Ph.D. U. of Alabama Sch. of Home Economics, University. *Dir., Research Lab. of Human Nutrition.* (5, 1949)

MEYER, KARL, M.D., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Biochem.* (2, 1934)

MEYER, KARL F., M.D., Ph.D. George W. Hooper Fndn., U. of California Med. Ctr., San Francisco. *Dir.* (4R, 1930; 6, 1922)

MEYER, ROLAND K., Ph.D. U. of Wisconsin, Birge Hall, Madison 6. *Marshall Prof. of Zool.* (1, 1949)

MEYERS, FREDERICK H., M.D. U. of California Sch. of Med., Dept. of Pharmacol., San Francisco 22. *Asst. Prof.* (3, 1955)

MEZEY, KALMAN, M.D. Calle 84, No. 9-68, Bogota, Colombia. (3, 1950)

MIALE, JOHN B., M.D. Jackson Mem. Hosp., Miami, Fla. *Dir., Clin. Pathol. Labs.; Prof. Pathol., U. of Miami.* (4, 1956)

MICHAELIS, MORITZ, Ph.D. U. of Maryland Sch. of Med., Dept. of Exptl. Surgery, Baltimore 1. *Research Assoc. in Surgery.* (2, 1958)

MICHEL, HARRY O., Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Chief, Bioanalytical Br.* (2, 1949)

MICKELSEN, OLAF, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Lab. of Nutrition and Endocrinol.* (2, 1944; 5, 1956)

MIDER, GEORGE BURROUGHS, M.D. Natl. Cancer

Inst., NIH, Bethesda 14, Md. *Assoc. Dir. in Charge of Research.* (4, 1940)

MIGICOVSKY, BERT B., Ph.D. Canada Dept. of Agric., Div. of Chem., Sci. Serv., Ottawa, Canada. *Head, Animal Chem.* (2, 1956)

MIHALYI, ELEMER, M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1954)

MIHICH, ENRICO, M.D. Roswell Park Mem. Inst., Dept. of Exptl. Therap., Buffalo 3, N.Y. *Sr. Cancer Research Scientist.* (3, 1958)

MILCH, LAWRENCE J., Ph.D. USAF Sch. of Aviation Med., Randolph AFB, Tex. *Chief, Dept. of Pharmacol. and Biochem.* (3, 1954)

MILES, WALTER R., Ph.D. U.S. Naval Med. Research Lab., U.S. Submarine Base, New London, Conn. *Sci. Dir.* (1, 1919)

MILHORAT, ADE T., M.D. Cornell U. Med. Coll., New York City 21. *Prof. Clin. Med.; Res. Assoc., Russell Sage Inst. Pathol.* (1, 1934; 3, 1937; 5, 1935)

MILITZER, WALTER E., Ph.D. U. of Nebraska, Lincoln. *Dean, Coll. of Arts & Sci.* (2, 1951)

MILLER, AUGUSTUS TAYLOR, JR., Ph.D., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Prof. of Physiol.* (1, 1944)

MILLER, BENJAMIN F., M.D. May Inst. for Med. Res., Jewish Hosp., Cincinnati 29, Ohio. *Dir.; Assoc. Prof. Exptl. Med., U. of Cincinnati.* (2, 1938)

MILLER, C. PHILLIP, M.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Prof.* (4, 1925; 6, 1928)

MILLER, CAREY D., M.S. U. of Hawaii, Honolulu. *Prof. of Foods and Nutrition, Hawaii Agric. Expt. Sta.* (5, 1942)

MILLER, ELIZABETH C., Ph.D. U. of Wisconsin, McArdle Mem. Lab. for Cancer Research, Madison 6. *Asst. Prof. of Oncol.* (2, 1951)

MILLER, FRANKLIN R., M.D. Snyder Clinic, 103½ E. 9th St., Winfield, Kans. (4, 1940)

MILLER, FREDERICK R., M.D. 280 Carlton St., Toronto 2, Canada. (1R, 1908)

MILLER, GAIL L., Ph.D. QM Research and Engineering Ctr., Natick, Mass. *Head, Biochem.* (2, 1949)

MILLER, GEORGE A., Ph.D. Harvard U., Psychol. Dept., Cambridge 38, Mass. *Prof.* (1, 1948)

MILLER, GEORGE H., M.D. Amer. Coll. of Surgeons, 40 E. Erie St., Chicago 11, Ill. *Asst. Dir.* (3, 1925)

MILLER, JACK W., Ph.D. U. of Wisconsin, Dept. of Pharmacol. and Toxicol., Madison 6. *Asst. Prof.* (3, 1956)

MILLER, JAMES A., Ph.D. U. of Wisconsin Med. Sch., McArdle Mem. Lab. for Cancer Research, Madison 6. *Prof. of Oncol.* (2, 1949)

MILLER, JAMES ALBERT, JR., Ph.D. Emory U., Atlanta 22, Ga. *Prof. of Anatomy.* (1, 1957)

MILLER, LEON L., Ph.D., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Assoc. Prof. Radiation Biol. & Biochem.* (2, 1947)

MILLER, LILA, Ph.D. U. of Michigan, Ann Arbor. *Assoc. Prof. of Biol. Chem.* (2, 1946)

MILLER, LLOYD C., Ph.D. U.S. Pharmacopeia, 46 Park Ave., New York City 16. *Dir. of Revision.* (3, 1938)

MILLER, O. NEAL, Ph.D. Tulane U. Sch. of Med., New Orleans, La. *Assoc. Prof. of Biochem. and Med.* (2, 1954; 5, 1956)

MILLER, R. C., Ph.D. Pennsylvania State U., University Park. *Prof. of Agric. and Biol. Chem.* (5, 1935)

MILLER, RUSSELL F., Ph.D. Virginia Polytech. Inst., Blacksburg. *Assoc. Prof. of Biochem. & Nutrition.* (5, 1958)

MILLER, RUTH E., Ph.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. *Prof. of Bacteriol.* (6, 1954)

MILLER, ZELMA BAKER, Ph.D. 511 Evanswood Pl., Cincinnati 20, Ohio. (2, 1940)

MILLICAN, R. CARL, M.D., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Med. Dir., PHS.* (3, 1951)

MILLICHAP, J. GORDON, M.D. Albert Einstein Coll. of Med., New York City 61. *Assoc. Prof. of Pharmacol. and Pediatrics.* (3, 1957)

MILLMAN, NATHAN, M.Sc., Ph.D. Bayberry Rd., Somerville, N.J. *Dir., Div. of Physiol. & Pharmacol., Ortho Research Fndn.* (1, 1950)

MILLS, CLARENCE A., Ph.D., M.D. Cincinnati Gen. Hosp., Cincinnati 29, Ohio. *Prof. of Exptl. Med., U. of Cincinnati.* (1, 1921; 2, 1921)

MILLS, LEWIS C., JR., M.D. Hahnemann Med. Coll., Philadelphia, Pa. *Assoc. Prof. of Med.* (3, 1957)

MILLS, RUSSELL C., Ph.D. U. of Kansas, Dept. of Biochem., Lawrence. *Prof.* (2, 1956)

MINARD, DAVID, Ph.D., M.D. 5401 Edgemoor Lane, Bethesda 14, Md. (1, 1950)

MINOT, ANN S., Ph.D. Vanderbilt U. Sch. of Med., Nashville, Tenn. *Prof. of Biochem.; Dir., Clin. Chem. Labs.* (1, 1923)

MINZ, BRUNO, M.D. Osawatomie State Hosp., Osawatomie, Kans. *Dir. of Res.; Prof. of Pharmacol., U. of Kansas Med. Ctr.* (3, 1952)

MIRICK, GEORGE S., M.D. Johns Hopkins U., Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Med.* (6, 1950)

MIRSKY, ALFRED E., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member.* (2, 1941)

MIRSKY, I. ARTHUR, M.D. U. of Pittsburgh Sch. of Med., Dept. of Clin. Sci., Pittsburgh 13, Pa. *Prof. and Chmn.* (1, 1936)

MITCHELL, HAROLD H., M.D. 9730 Wilshire Blvd., Beverly Hills, Calif. *Assoc. Scientist, Rand Corp.* (6, 1943)

MITCHELL, HAROLD H., Ph.D. U. of Illinois, Div. of Animal Nutrition, Urbana. *Prof. Emer.* (2R, 1919; 5, 1933)

MITCHELL, HELEN S., Ph.D. U. of Massachusetts, Amherst. *Dean, Sch. of Home Economics.* (5, 1933)

MITCHELL, HERSCHEL K., Ph.D. California Inst. of Technology, Biol. Div., Pasadena. *Prof.* (2, 1955)

MITOMA, CHOZO, Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1957)

MIXNER, JOHN PAULDING, Ph.D. Rutgers U. Dairy Res. Farm, Sussex, N.J. *Prof. of Dairy Husbandry; Res. Spec. in Dairy Husbandry.* (1, 1958)

MIYA, TOM S., Ph.D. Purdue U., Dept. of Pharmacol., Lafayette, Ind. *Prof.* (3, 1956)

MODELL, WALTER, M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Pharmacol. (Clin. Pharmacol.)* (3, 1944)

MOLNAR, GEORGE W., Ph.D. USA Med. Research New York Sch. of Med., Dept. of Physiol., Syracuse. *Prof. and Head of Dept.* (1, 1952)

MOHN, JAMES F., M.D. U. of Buffalo Sch. of Med., Dept. of Bacteriol. and Immunol., Buffalo 14, N.Y. *Assoc. Prof.* (6, 1946)

MOLDAVE, KIVIE, Ph.D. Tufts U. Sch. of Med., Dept. of Biochem., Boston 11, Mass. *Assoc. Prof.* (2, 1957)

MOLITOR, HANS, M.D. Merck Sharp & Dohme Research Labs., Rahway, N. J. *Dir., Sci. Relations.* (1, 1933; 3, 1942)

MOLLAND, JACOB, M.D., Ph.D. U. of Oslo, Dept. of Pharmacol., Oslo, Norway. *Prof. and Head of Dept.* (3, 1948)

MOE, GORDON KENNETH, Ph.D., M.D. State U. of Lab., Fort Knox, Ky. *Physiologist.* (1, 1949)

MOLOMUT, NORMAN, Ph.D. Waldemar Med. Research Fndn., Port Washington, N.Y. *Dir. of Research.* (6, 1942)

MOMMAERTS, W. F. H. M., Ph.D. U. of California Med. Ctr., Los Angeles. *Prof. Med. & Physiol.; Dir., Cardio-Vasc. Res. Lab.* (1, 1955; 2, 1951)

MONGE, CARLOS, D.Sc.(Hon.) Natl. Inst. of Andean Biol., Lima, Peru. *Dir., School of Advanced Studies.* (1H, 1952)

MONKHOUSE, FRANK C., Ph.D. U. of Toronto, Dept. of Physiol., Toronto, Canada. *Assoc. Prof.* (1, 1955)

MONTGOMERY, ARTHUR V., JR., M.D., Ph.D. U. of Colorado, Dept. of Surgery (Research), Denver 20. *Instr.* (1, 1955)

MONTGOMERY, HUGH, M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Assoc. Prof. of Med.* (1, 1950)

MONTGOMERY, REX, Ph.D. State U. of Iowa, Dept. of Biochem., Iowa City. *Asst. Prof.* (2, 1957)

MONTO, RAYMOND W., M.D. Henry Ford Hosp., Detroit 2, Mich. *Physician-in-Charge of Hematol.* (4, 1958)

MOON, HENRY D., M.D. U. of California Sch. of Med., Dept. of Pathol., San Francisco 22. *Chmn.; City Pathologist.* (4, 1950)

MOON, VIRGIL H., M.Sc., M.D. 824 Majorca Ave., Coral Gables 34, Fla. *Prof. Emer. of Pathol., U. of Miami.* (4R, 1934)

MOORE, A. R., Ph.D. 133 Third St., Pacific Grove, Calif. (1R, 1912)

MOORE, ALICE E., M.D. Sloan-Kettering Inst., New York City 21. *Assoc.; Assoc. Prof., Sloan-Kettering Div., Cornell U.* (6, 1956)

MOORE, CARL VERNON, M.D. Washington U. Sch. of Med., St. Louis, Mo. *Prof. of Med.* (4, 1938; 5, 1941)

MOORE, DAN H., Ph.D. Rockefeller Inst., 66th St. at York Ave., New York City 21. (1, 1948)

MOORE, JAMES C., M.D. U. of Louisville Sch. of Med., Louisville 2, Ky. *Assoc. Prof. of Physiol.* (1, 1957)

MOORE, LANE A., Ph.D. USDA, Dairy Cattle Research Br., Beltsville, Md. *Chief.* (5, 1940)

MOORE, RICHARD D., M.D. Western Reserve U. Inst. of Pathol., Cleveland 6, Ohio. (4, 1957)

MOORE, ROBERT A., M.D., Ph.D. State U. of New York, Brooklyn 3. *Pres., Downstate Med. Ctr.* (4, 1929)

MOORE, STANFORD, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1949)

MOORHOUSE, VICTOR H. K., M.D. 32 Amanda St., Orangeville, Ont., Canada. *Prof. Emer. Physiol., U. of Manitoba.* (1R, 1912)

MORALES, MANUEL F., Ph.D. Dartmouth Med. Sch., Dept. of Biochem., Hanover, N. H. *Chmn. of Dept.* (1, 1950)

MORAN, NEIL C., M.D. Emory U., Div. of Basic Health Sci., Dept. of Pharmacol., Atlanta 22, Ga. *Assoc. Prof.* (3, 1956)

MORAN, THOMAS J., M.D. U. of Pittsburgh Sch. of Med., Dept. of Pathol., Pittsburgh 13, Pa. *Prof.* (4, 1955)

MORE, ROBERT H., M.Sc., M.D. Queen's U., Dept. of Pathol., Kingston, Canada. *Prof. and Head of Dept.* (4, 1949)

MOREHOUSE, LAURENCE E., Ph.D. U. of California, Dept. of Physical Educ., Los Angeles 24. *Prof.* (1, 1947)

MOREHOUSE, MARGARET G., Ph.D. U. of Southern California Sch. of Med., Los Angeles 7. *Assoc. Prof. of Biochem.* (2, 1952)

MORELL, SAMUEL A., Ph.D. Pabst Labs., Milwaukee, Wis. *Head, Organic and Fine Chem. Lab.* (2, 1954)

MORGAN, AGNES FAY, Ph.D. U. of California, Berkeley 4. *Prof. Emer. of Nutrition; Biochemist, Agric. Expt. Sta.* (2R, 1929; 5, 1933)

MORGAN, CHARLES F., Ph.D. Georgetown U. Sch. of Med. and Dent., Dept. of Physiol., Washington 7, D.C. *Prof. and Chmn.* (1, 1948; 3, 1947)

MORGAN, CLIFFORD T., Ph.D. Town Point, Cambridge, Md. (1, 1943)

MORGAN, COUNCILMAN, M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. in Microbiol. and Med.* (6, 1956)

MORGAN, HERBERT R., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N. Y. *Prof. of Bacteriol.* (6, 1957)

MORGAN, JOSEPH F., Ph.D. Dept. of Natl. Health and Welfare, Lab. of Hygiene, Ottawa, Canada. *Chief, Biochem. Res. Labs.* (2, 1953)

MORGULIS, SERGIUS, Ph.D. U. of Nebraska Coll. of Med., Omaha 5. *Prof. Emer.* (1R, 1914; 2R, 1916)

MORIN, F. A., M.D., Ph.D. Wayne State U. Coll. of Med., Dept. of Anatomy, Detroit 7, Mich. *Prof.* (1, 1954)

MORISON, ROBERT S., M.D. Rockefeller Fndn., 49 W. 49th St., New York City 20. *Dir. for Biol. and Med. Research.* (1, 1938)

MORITZ, ALAN R., M.D. Western Reserve U. Inst. of Pathol., Cleveland 6, Ohio. *Prof. of Pathol. and Inst. Dir.* (4, 1934)

MORRELL, CLARENCE ALLISON, Ph.D. Dept. of Natl. Health and Welfare, Ottawa, Canada. *Dir., Food & Drug Directorate.* (3, 1937)

MORRIS, DAVID M., Ph.D. U. of Miami Med. Br., Anatomy Dept., Coral Gables, Fla. *Asst. Prof.* (1, 1957)

MORRIS, HAROLD P., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Nutr. & Carcinogen. Sec., Biochem. Lab.* (2, 1944; 5, 1943)

MORRIS, J. ANTHONY, M.Sc., Ph.D. 406th Med. Gen. Lab., APO 343, San Francisco, Calif. *Asst.*

Chief. (6, 1951)

MORRIS, LUCIEN E., M.D. U. of Washington Sch. of Med., Seattle 5. *Prof. of Anesthesiol.* (3, 1952)

MORRIS, MARION C., Ph.D. Natl. Fndn. for Infantile Paralysis, 301 E. 42nd St., New York City 17. *Asst. Dir., Professional Educ.* (6, 1936)

MORRISON, ALEXANDER B., Ph.D. Mead Johnson & Co., Res. Labs., Evansville 21, Ind. *Assoc. Sr. Chemist.* (5, 1958)

MORRISON, DEMPSIE B., Ph.D. U. of Tennessee Coll. of Med., Memphis. *Dir. of Clin. Chem. and Prof. of Chem.* (2, 1936)

MORRISON, MARTIN, Ph.D. U. of Rochester Med. Ctr., Dept. of Biochem., Rochester 20, N.Y. *Asst. Prof.* (2, 1957)

MORRISON, PETER R., Ph.D. U. of Wisconsin, Madison 6. *Assoc. Prof. of Zool. and Physiol.* (1, 1952; 2, 1950)

MORROW, PAUL EDWARD, Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Asst. Prof.* (3, 1958)

MORSE, MINERVA, Ph.D. 5525 Kimbark Ave., Chicago, Ill. (2, 1934)

MORTIMER, BERNARD, Ph.D., M.D. 25 N. Ottawa St., Joliet, Ill. *Silver Cross and St. Joseph's Hosps.* (1, 1936)

MORTON, JOHN J., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. Emer. of Surgery.* (4R, 1927)

MOSBACH, E. H., Ph.D. Columbia U. Res. Serv., Goldwater Mem. Hosp., Welfare Is., New York City 17. *Asst. Prof. Biochem.* (2, 1957)

MOSES, CAMPBELL, M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Assoc. Prof. of Med.* (1, 1948)

MOSKOWITZ, MERWIN, Ph.D. Purdue U., Dept. of Biol. Sci., Lafayette, Ind. *Assoc. Prof. of Bacteriol.* (6, 1957)

MOSS, W. GLEN, Ph.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Prof.* (1, 1951)

MOTLEY, HURLEY L., Ph.D., M.D. 1212 Shatto St., Los Angeles 17, Calif. *Prof. of Med. and Dir., Cardio-Resp. Lab., U. of Southern Calif.* (1, 1947)

MOULDER, JAMES W., Ph.D. U. of Chicago, Depts. of Microbiol. and Biochem., Chicago 37, Ill. *Prof. of Microbiol.* (2, 1950)

MOUNTAIN, ISABEL M., Ph.D. Babies Hosp., 3975 Broadway, New York City 32. (6, 1947)

MOUNTCASTLE, VERNON B., M.D. Johns Hopkins U. Sch. of Med., Dept. of Physiol, Baltimore 5, Md. *Assoc. Prof. of Physiol.* (1, 1949)

MOUNTER, L. A., Ph.D. Med. Coll. of Virginia, Dept. of Biophysics, Richmond. *Assoc. Prof.* (2, 1956)

MOXON, ALVIN L., Ph.D. Ohio Agric. Expt. Sta., Wooster. *Prof., Animal Sci. and Biochem., Ohio State U.* (2, 1944)

MOYED, HARRIS S., Ph.D. Harvard Med. Sch., Dept. of Bacteriol., Boston 15, Mass. *Instr.* (2, 1958)

MOYER, ARDEN W., Ph.D. 138 Fremont Ave., Park Ridge, N.J. *Research Biochemist, Amer. Cyanamide Co., Lederle Labs.* (6, 1946)

MOYER, CARL A., M.D. Washington U. Sch. of Med., Dept. of Surgery, St. Louis 10, Mo. (1, 1943)

MOYER, ELSIE Z., M.S. USDA, Human Nutrition Res. Div., ARS. Washington 25, D.C. *Nutrition Specialist.* (5, 1958)

MOYER, JOHN HENRY, III, M.D. Hahnemann Med. Coll. and Hosp., Philadelphia, Pa. *Prof. of Med.* (3, 1951)

MUDD, STUART, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Microbiol.* (1, 1921; 4, 1927; 6, 1927)

MUDGE, GILBERT H., M.D., D.M.S. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Prof. of Pharmacol.* (1, 1951; 3, 1956)

MUEHLBERGER, CLARENCE W., Ph.D. State Health Dept. Labs., Lansing 4, Mich. *State Toxicologist.* (3, 1928)

MUELLER, GERALD C., M.D., Ph.D. U. of Wisconsin, McArdle Lab., Madison. *Assoc. Prof.* (2, 1952)

MUIRHEAD, E. E. U. of Texas Southwestern Med. Sch., Dept. of Pathol., Dallas. *Prof. and Hematologist.* (1, 1953; 4, 1952)

MUKERJI, B., D.Sc., M.B. Central Drug Research Inst., Lucknow, India. *Dir.* (3, 1938)

MULFORD, DWIGHT J., Ph.D. U. of Kansas Sch. of Med., Dept. of Biochem., Lawrence. *Prof.* (2, 1948)

MULINOS, M. G., M.D., Ph.D. Commercial Solvents Corp., 260 Madison Ave., New York City 16. *Med. Research Dir.* (3, 1931)

MULL, JAMES W., Ph.D. 1870 Grove Ave., Quincy, Ill. *Assoc., Quincy Specialties Co.* (2, 1937)

MULLER, OTTO H., Dr. rer. nat. State U. of New York, Upstate Med. Ctr., Dept. of Physiol., Syracuse 10. *Prof.* (1, 1947)

MULLER, ROBERT H., M.D. 36 Glen Ridge Pkwy., Montclair, N.J. *Internist, Microbiologist.* (6, 1950)

MULLIGAN, RICHARD M., M.D. U. of Colorado Sch. of Med., Denver 20. *Prof. of Pathol.* (4, 1947)

MULLIN, F. J., Ph.D. Shimer Coll., Mount Carroll, Ill. *Pres.* (1, 1937)

MULLINS, L. J., Ph.D. Purdue U., Biophys. Lab., Lafayette, Ind. *Assoc. Prof. of Biophysics.* (1, 1952)

MUNOZ, JOHN J., Ph.D. Montana State U., Missoula. *Prof. of Bacteriol.* (6, 1951)

MUNSELL, HAZEL E., Ph.D. 25 Audubon St., New Britain, Conn. (5, 1933)

MUNSON, PAUL L., Ph.D. Harvard Sch. of Dental Med., Boston 15, Mass. *Assoc. Prof. of Pharmacol.* (2, 1950; 3, 1954)

MUNTWYLER, EDWARD, Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Prof. of Biochem.* (2, 1931)

MUNTZ, JOHN A., Ph.D. Albany Med. Coll., Dept. of Biochem., Albany 8, N.Y. *Prof. and Chmn. of Dept.* (2, 1948)

MURLIN, JOHN R., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. Emer. of Physiol.* (1R, 1906; 2R, 1908; 5R, 1933)

MURPHY, GEORGE E., M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Pathol.* (4, 1954)

MURPHY, QUILLIAN R., M.D., Ph.D. U. of Wisconsin Med. Sch., Dept. of Physiol., Madison 6. *Assoc. Prof.* (1, 1949)

MURRAY, EVERITT G. D., M.D., D.Sc. U. of Western Ontario, Dept. of Med. Research, London, Can-

ada. *Research Prof.* (6, 1933)

MURRAY, F. JOSEPH, Ph.D. The Wm. S. Merrell Co., Lockland Sta., Cincinnati, Ohio. *Asst. to Vice-Pres. for Research.* (6, 1950)

MURRAY, RODERICK, M.D. Natl. Insts. of Health, Div. of Biologics Standards, Bethesda 14, Md. *Dir.* (6, 1958)

MURRAY, T. KEITH, Ph.D. Dept. of Health and Welfare, Ottawa, Canada. *Chemist.* (5, 1958)

MUSACCHIA, X. J., Ph.D. St. Louis U. Biol. Labs., 1402 S. Grand Blvd., St. Louis 4, Mo. *Assoc. Prof.* (1, 1952)

MUSCHEL, LOUIS H., Ph.D. Walter Reed Army Inst. of Research, Dept. of Serol., Washington 12, D.C. *Chief.* (6, 1956)

MUSHETT, CHARLES W., Ph.D. Merck Sharp & Dohme Research Labs., Rahway, N.J. *Asst. Dir., Scientific Relations.* (4, 1948)

MUUS, JYTTE, Mag. Scient. Mount Holyoke Coll., South Hadley, Mass. *Prof. of Biochem.* (2, 1946)

MYERS, J. D., M.D. U. of Pittsburgh Sch. of Med., Dept. of Med., Pittsburgh 13, Pa. *Prof.* (1, 1951)

MYERS, WILLIAM G., Ph.D., M.D. 2724 Wexford Rd., Columbus 21, Ohio. *Stone Research Prof. of Med. Biophysics, Ohio State U.* (1, 1952)

MYRVIK, QUENTIN N., Ph.D. U. of Virginia Sch. of Med., Charlottesville. *Asst. Prof. of Microbiol.* (6, 1954)

NACHMANSOHN, DAVID, M.D. Columbia U. Coll. of P. & S., Dept. of Neurol., New York City 32. *Prof. of Biochem.* (1, 1940; 2, 1951)

NADEL, ELI M., Natl. Insts. of Health, Bethesda 14, Md. *Patbologist.* (4, 1953)

NADLER, J. ERNEST, M.D., Med.D.Sc. 80-16 Lefferts Blvd., Kew Gardens 15, N.Y. *Asst. Prof. Med., New York Med. Coll.* (3, 1940)

NAHAS, GABRIEL G., M.D., Ph.D. Walter Reed Army Inst. of Res., Dept. of Cardio-Resp. Dis., Washington 12, D.C. *Chief, Resp. Sec.* (1, 1957)

NAHUM, LOUIS H., M.D. Yale Med. Sch., New Haven 11, Conn. *Lect.* (1, 1934)

NAJJAR, VICTOR A., M.D. Vanderbilt U. Sch. of Med., Dept. of Microbiol., Nashville 5, Tenn. *Prof. and Head of Dept.* (2, 1946)

NAKADA, HENRY I., Ph.D. 476 Prospect St., La Jolla, Calif. *Member, Research Staff, Scripps Clinic and Research Fndn.* (2, 1957)

NASH, JOE BERT, Ph.D. U. of Texas Med. Br., Dept. of Pharmacol. and Toxicol., Galveston. *Assoc. Prof.* (3, 1958)

NASH, THOMAS P., JR., Ph.D. 62 So. Dunlap St., Memphis, Tenn. *Dean, Sch. of Biol. Sci., U. of Tennessee Coll. of Med.* (2, 1923)

NASON, ALVIN, Ph.D. Johns Hopkins U., McCollum-Pratt Inst., Baltimore 18, Md. *Prof.* (2, 1953)

NASSET, EDMUND S., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. of Physiol.* (1, 1932; 5, 1940)

NASTUK, WILLIAM L., Ph.D. Columbia U., Coll. of P. & S., Dept. of Physiol., New York City 32. *Assoc. Prof.* (1, 1949)

NATELSON, SAMUEL, Ph.D. Roosevelt Hosp., Dept. of Biochem., New York City 19. *Dept. Head.* (2, 1950)

NECHELES, HEINRICH, M.D., Ph.D. Michael Reese Hosp., Chicago, Ill. *Dir. of Gastrointestinal Research.* (1, 1929)

NEFF, WILLIAM D., Ph.D. U. of Chicago, Dept. of Psychol., Faculty Exchange, Chicago 37, Ill. *Prof.* (1, 1950)

NEILANDS, JOHN B., Ph.D. U. of California, Dept. of Biochem., Berkeley. *Assoc. Prof.* (2, 1953)

NEILL, JAMES M., Ph.D. Cornell U. Med. Coll., New York City 21. *Prof. of Bacteriol. and Immunol.* (6, 1930)

NEILSON, CHARLES HUGH, Ph.D., M.D. Humboldt Bldg., St. Louis, Mo. *Physician and Internist, St. John's Hosp.* (2, 1906)

NELSON, ARTHUR A., M.D., Ph.D. 1714 Corwin Dr., Silver Spring, Md. *Med. Officer (Pathol.), Food & Drug Admin., Div. of Pharmacol.* (4, 1942)

NELSON, CARL T., M.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Dermatol.* (6, 1943)

NELSON, DOROTHY, Ph.D. U. of Illinois Coll. of Med., Dept. of Clin. Sci., Chicago. *Research Assoc.* (1, 1956)

NELSON, E. M., Ph.D. Food and Drug Admin., Dept. of HEW, Washington 25, D.C. (2, 1927; 5, 1933)

NELSON, ERIC L., Ph.D. U. of California Med. Ctr., Dept. of Infectious Diseases, Los Angeles 24. (4, 1957)

NELSON, ERWIN E., Ph.D., M.D. U. of New Mexico, Albuquerque. *Visting Lect. in Pharmacol.* (3R, 1924)

NELSON, JOHN B., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Member.* (4, 1934)

NELSON, JOHN M., Ph.D. 39 Claremont Ave., New York City 27. *Prof. Emer. Organic Chem., Columbia U.* (2R, 1923)

NELSON, MARJORIE M., Ph.D. U. of California, Inst. of Exptl. Biol., Berkeley. *Assoc. Research Biochemist.* (5, 1950)

NELSON, NORTON, Ph.D. New York U. Med. Ctr., New York City 16. *Prof. of Indust. Med.* (2, 1946)

NELSON, P. MABEL, Ph.D. 1829 Santa Ana Ave., Costa Mesa, Calif. (5R, 1934)

NELSON, WALTER L., Ph.D. Cornell U., Dept. of Biochem., Ithaca, N.Y. *Prof.* (2, 1956; 5, 1951)

NELSON, WARREN O., Ph.D., M.D. Rockefeller Inst., Population Council, New York City 21. *Med. Dir.* (1, 1937)

NETER, ERWIN, M.D. Children's Hosp., 219 Bryant St., Buffalo, N.Y. *Dir., Dept. of Bacteriol.* (6, 1937)

NETTLESHIP, ANDERSON, M.D. Fayetteville, Ark. (4, 1942)

NEUMAN, WILLIAM F., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Assoc. Prof. of Biochem. and Pharmacol.* (2, 1949; 3, 1952)

NEUMANN, CHARLES G., M.D. 2 Schuyler St., New Rochelle, N.Y. *Assoc. Prof. of Clin. Surgery, N.Y.U. Post-Grad. Med. Sch.* (1, 1944)

NEURATH, HANS, Ph.D. U. of Washington Sch. of Med., Dept. of Biochem., Seattle 5. *Prof. and Exec. Officer of Dept.* (2, 1940; 6, 1944)

NEUWIRTH, ISAAC, Ph.D. 209 E. 23rd St., New York City 10. *Prof. of Pharmacol., New York U. Coll. of Dent.* (2, 1924; 3, 1931)

NEWBURGH, ROBERT W., Ph.D. Science Research

Inst., Oregon State Coll., Corvallis. *Assoc. Prof. of Chem.; Chemist, USDA.* (2, 1957)

NEWMAN, ELLIOT V., M.D. Vanderbilt U. Hosp., Nashville, Tenn. *Prof. of Exptl. Med.* (1, 1948)

NEWMAN, HENRY W., M.D. Stanford U. Sch. of Med., San Francisco 15, Calif. *Prof. of Med.* (3, 1949)

NEWMARK, PHILIP, Ph.D. U. of Kansas, Dept. of Biochem., Lawrence. *Asst. Prof.* (2, 1958)

NGAI, SHIH-HSUN, M.B. Columbia U. Coll. of P. & S., New York City 32. *Asst. Prof. of Anesthesiol.* (3, 1958)

NICE, LEONARD B., Ph.D. 5725 S. Harper Ave., Chicago 37, Ill. *Prof. Emer. of Physiol., Chicago Med. Sch.* (1, 1921)

NICHOL, CHARLES A., Ph.D. Roswell Park Mem. Inst., Buffalo 3, N.Y. *Dir. of Exptl. Therap.* (2, 1954)

NICHOLAS, JOHN S., Ph.D. Yale U., Osborn Zool. Lab., New Haven, Conn. *Sterling Prof. of Biol.* (1, 1927)

NICHOLS, JOHN M., Ph.D., M.D. U. of Kansas Sch. of Med., Dept. of Pathol., Kansas City. *Asst. Prof.* (4, 1957)

NICHOLSON, HAYDEN C., M.D. Hosp. Council of Greater New York, 3 E. 54th St., New York City 22. *Exec. Dir.* (1, 1932)

NICKERSON, JOHN L., Ph.D. Chicago Med. Sch., Chicago 12, Ill. *Prof. and Chmn., Physiol. and Pharmacol.* (1, 1945)

NICKERSON, MARK, Ph.D., M.D. U. of Manitoba Med. Sch., Dept. of Pharmacol. & Therap., Winnipeg, Canada. *Prof. and Head.* (1, 1951; 3, 1947)

NICKERSON, WALTER J., Ph.D. Rutgers, The State U., Inst. of Microbiol., New Brunswick, N. J. *Prof. of Microbiol. Biochem.* (2, 1958)

NICOLL, PAUL A., Ph.D. Indiana U., Bloomington. *Prof. of Physiol.* (1, 1945)

NIEMANN, CARL G., Ph.D. California Inst. of Tech., Pasadena 4. *Prof. of Organic Chem.* (2, 1940)

NIMS, LESLIE F., Ph.D. Brookhaven Natl. Lab., Biol. Dept., Upton, N.Y. *Sr. Physiologist.* (1, 1940)

NISHIMURA, EDWIN T., M.D. Atomic Bomb Casualty Comm., APO 354, San Francisco, Calif. *Patbologist.* (4, 1956)

NISONOFF, ALFRED, Ph.D. Roswell Park Mem. Inst., Buffalo 3, N.Y. *Assoc. Cancer Res. Scientist.* (2, 1958)

NITOWSKY, HAROLD M., M.D. Sinai Hosp of Baltimore, Inc., Baltimore 5, Md. *Head, Pediatrics Res.* (5, 1958)

NOBLE, ROBERT LAING, M.D., Ph.D., D.Sc. U. of Western Ontario, Dept. of Med. Research, London, Canada. (1, 1941)

NODA, LAFAYETTE, Ph.D. Dartmouth Med. Sch., Dept. of Biochem., Hanover, N.H. *Assoc. Prof.* (2, 1955)

NOELL, WERNER K., M.D. Roswell Park Mem. Inst. Buffalo 3, N.Y. *Prin. Research Scientist.* (1, 1950)

NOLL, HANS, Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Microbiol., Pittsburgh 13, Pa. (2, 1958)

NOONAN, THOMAS R., M.D. U. of Rochester, Dept. of Radiation Biol., Rochester 20, N.Y. *Assoc. Prof.* (1, 1955)

NORD, F. F., D.Sc. Fordham U., New York City 58. *Prof. of Organic Chem. and Enzymol.* (2, 1940)

NORMAN, TOM D., M.D. U. of Arkansas Med. Ctr., Dept. of Pathol., Little Rock. *Assoc. Prof.* (4, 1957)

NORRIS, L. C., Ph.D. Cornell U., Rice Hall, Ithaca, N.Y. *Prof. of Nutrition.* (2, 1939; 5, 1934)

NORRIS, WILLIAM P., Ph.D. Argonne Natl. Lab., Div. of Biol. and Med. Res., Lemont Ill. *Assoc. Biochemist.* (2, 1957)

NORTH, WILLIAM C., M.D., Ph.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Asst. Prof. of Pharmacol.* (3, 1955)

NORTHROP, J. H., Ph.D., Sc.D. U. of California, Berkeley 4. *Member of Rockefeller Inst.* (2, 1938)

NORTHUP, DAVID W., Ph.D. West Virginia U. Sch. of Med., Dept. of Physiol., Morgantown. *Prof. and Head of Dept.* (1, 1936)

NORTON, STATA, Ph.D. Wellcome Research Labs., Tuckahoe, N.Y. *Pharmacologist.* (3, 1955)

NOTHMAN, MARTIN M., M.D. New England Ctr. Hosp., Boston 11, Mass. *Asst. Clin. Prof. of Int. Med.* (3, 1954)

NOVELLI, G. DAVID, Ph.D. Oak Ridge Natl. Lab., Biol. Div., P.O. Box Y, Oak Ridge, Tenn. (2, 1951)

NOVIKOFF, ALEX B., Ph.D. Albert Einstein Coll. of Med., New York City 61. (2, 1952)

NOWINSKI, W. W., Ph.D. U. of Texas Sch. of Med., Galveston. *Assoc. Prof. Biochem.; Dir., Tissue Metab. Res. Lab.* (1, 1948)

NUNGESTER, WALTER JAMES, Sc.D., M.D. U. of Michigan, Dept. of Bacteriol., Ann Arbor. *Prof. and Chmn. of Dept.* (6, 1949)

NYBOER, JAN, Sc.D., M.D. Harper Hosp., 3825 Brush, Detroit 1, Mich. (1, 1951)

NYC, JOSEPH F., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Physiol. Chem.* (2, 1952)

OBERST, FRED W., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Chief, Gassing Br.* (2, 1936; 3, 1949)

OCHOA, SEVERO, M.D. New York U. Coll. of Med., Dept. of Biochem., New York City 16. *Prof. and Chmn. of Dept.* (2, 1942)

O'DELL, BOYD L., Ph.D. U. of Missouri, 105 Schweitzer Hall, Columbia. *Prof. of Agric. Chem.* (2, 1952; 5, 1953)

ODELL, THEODORE T., JR., Ph.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Biologist.* (1, 1957)

O'DELL, THOMAS B., Ph.D. Irwin, Neisler Research Labs., Pharmacol. Sec., Decatur, Ill. *Dir., Biol. Research Sec.* (3, 1956)

ODLAND, LURA M., Ph.D. USDA, State Expt. Sta. Div., Washington 25, D.C. *Nutritionist.* (5, 1956)

OESPER, PETER, Ph.D. Hahnemann Med. Coll, Div. of Biochem., Philadelphia 2, Pa. *Assoc. Prof.* (2, 1956)

OESTER, Y. T., Ph.D., M.D. Loyola U., Stritch Sch. of Med., Chicago 12, Ill. *Prof. of Pharmacol.; Acting Chmn.* (3, 1954)

OESTERLING, M. JANE, Ph.D. Woman's Med. Coll. of Pennsylvania, Philadelphia 29. *Asst. Prof. of Physiol. Chem.* (2, 1958)

OGATA, TOMIO, M.D., D.M.Sc. U. of Tokyo, Faculty

of Med., Hongo, Tokyo, Japan. *Prof. of Serol.* (6, 1957)

OGDEN, ERIC. Ohio State U. Coll. of Med., Columbus 10. *Prof. of Physiol.* (1, 1941)

OGINSKY, EVELYN L., Ph.D. U. of Oregon Med. Sch., Dept. of Bacteriol., Portland 1. *Assoc. Prof.* (2, 1954)

OGLE, KENNETH N., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant, Biophysic. Res. & Ophthal.; Prof. Physiol. Optics, U. of Minn.* (1, 1953)

O'HARE, JAMES P., M.D. 520 Commonwealth Ave., Boston, Mass. *Physician, Peter Bent Brigham Hosp.; Emer. Lect. in Med., Harvard.* (4R, 1927)

OHLER, EDWIN ALLEN, Ph.D. Temple U. Sch. of Med., Dept. of Physiol., Philadelphia 40, Pa. *Assoc. Prof.* (1, 1954)

OHLSON, MARGARET A., Ph.D. State U. of Iowa, University Hosps., Iowa City. *Dir., Nutrition Dept.* (5, 1945)

OKEY, RUTH, Ph.D. U. of California, Dept. Nutrition & Home Econ., Berkeley 4. *Prof.; Biochemist, Agric. Expt. Sta.* (2, 1922; 5, 1933)

OKITA, GEORGE T., Ph.D. U. of Chicago, Dept. of Pharmacol., Chicago 37, Ill. *Asst. Prof.* (3, 1956)

OLCOTT, HAROLD S., Ph.D. U. of California, Dept. of Food Technol., Berkeley 4. *Prof. of Marine Food Technol.* (2, 1935)

OLDHAM, HELEN, Ph.D. USDA Research Ctr., Room 126 North Bldg., Beltsville, Md. *Nutrition Specialist.* (5, 1946)

O'LEARY, JOHN F., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Pharmacol. Br., Army Chemical Center, Md. *Asst. Br. Chief.* (3, 1957)

OLESON, J. J., Ph.D. Chas. Pfizer & Co., Inc., Maywood, N. J. (2, 1950)

OLITSKY, PETER K., M.D. Rockefeller Inst. for Med. Research, New York City 21. *Emer. Member.* (6R, 1917)

OLIVER, JEAN REDMAN, M.D. Buxton Rd., Chatham, N. J. *Distinguished Serv. Prof. Emer. of Pathol., N. Y. State U. Med. Coll.* (1, 1924; 4, 1924)

OLIVER, WADE W., M.D. 2749 N. E. 25th St., Lighthouse Pt., Pompano Beach, Fla. *Rockefeller Fndn., Assoc. Dir. (ret.)* (4R, 1925)

OLSEN, NORMAN S., Ph.D. Masonic Med. Research Lab., Utica, N. Y. *Asst. Dir.* (1, 1948; 2, 1949)

OLSON, BYRON J., Ph.D., M.D. PHS, Div. Foreign Quarantine, Dept. of HEW, Washington 25, D. C. *Chief, Epidemiol. and Immunization Br.* (6, 1948)

OLSON, CARL, JR., D.V.M., Ph.D. U. of Wisconsin, Dept. of Veterinary Sci., Madison. (4, 1937)

OLSON, JAMES ALLEN, Ph.D. U. of Florida Coll. of Med., Dept. of Biochem., Gainesville. *Asst. Prof.* (2, 1957)

OLSON, OSCAR E., Ph.D. 629-13th Ave., Brookings, S. D. *Head, Sta. Biochem., Agric. Expt. Sta., South Dakota State Coll.* (2, 1953)

OLSON, R. E., Ph.D., M.D. U. of Pittsburgh Grad. Sch., Dept. Biochem. & Nutr., Pittsburgh 13, Pa. *Prof. & Head.* (2, 1951; 5, 1952)

OLSON, RODNEY A., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Physiologist.* (1, 1953)

OLSON, WILLIAM H., M.D. Indiana State Prison, P. O. Box 41, Michigan City. *Physician-in-Charge.* (1, 1951)

OMACHI, AKIRA, Ph.D. U. of Illinois Coll of Med., Chicago 12. *Asst. Prof. of Physiol.* (1, 1955)

ONCLEY, J. L., D.Sc., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Prof. of Biol. Chem.* (2, 1955)

O'NEAL, ROBERT M., M.D. Washington U. Sch. of Med., St. Louis 10, Mo *Asst. Prof. of Pathol.* (4, 1956)

OPDYKE, DAVID F., Ph.D. Seton Hall Coll. of Med. & Dent., Dept. of Physiol., Jersey City 4, N. J. *Prof. and Chmn. of Dept.* (1, 1945)

OPIE, EUGENE L., M.D., Sc.D. Rockefeller Inst. for Med. Research, New York City 21. (1R, 1906; 4R, 1913; 6R, 1923)

OPPENHEIMER, ENID TRIBE. 1010 Fifth Ave., New York City 28. *Volunteer Research Worker, Inst. of Cancer Research, Columbia U.* (1, 1932)

OPPENHEIMER, ERNST, M.D. 199 Greenwood Way, Mill Valley, Calif. (3, 1944)

OPPENHEIMER, MORTON JOSEPH, Ed.M., M.D. Temple U. Sch. of Med., Philadelphia 40, Pa. *Prof. of Physiol.* (1, 1942)

OPSAHL, JEANETTE CLARA, Ph.D., M.D. Peter Bent Brigham Hosp., Boston 15, Mass. (2, 1952)

ORBELI, L. A. Academy of Sciences of the USSR, Moscow, USSR. (1H, 1946)

ORBISON, J. LOWELL, M.D. U. of Rochester, Rochester 20, N. Y. *Prof. of Pathol.* (4, 1955)

ORCUTT, JAMES A., Ph.D. Los Angeles County Air Pollution Control Dist., Los Angeles, Calif. (3, 1952)

ORKIN, LOUIS R., M.D. Albert Einstein Coll. of Med., Dept. of Anesthesiol., New York City 61. *Prof. and Chmn. of Dept.* (3, 1955)

ORLOFF, JACK, M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Dep. Chief, Lab. of Kidney and Electrolyte Metab. and Physiol.* (1, 1953)

ORMSBY, ANDREW A., Ph.D. U. of Texas Med. Br., Dept. of Biochem. and Nutrition, Galveston. *Prof. and Chmn. of Dept.* (2, 1954)

ORTEGA, LOUIS G., M.D. Mem. Ctr. for Cancer & Allied Diseases, New York City 21. *Asst. Attending Pathologist.* (4, 1958)

ORTEGA, PAUL, M.D. 6600 Lindenhurst Ave., Los Angeles, Calif. *Patbol. Staff, VA Hosp.; Clin. Patbol. Staff, UCLA Med. Sch.* (4, 1957)

ORTEN, ALINE UNDERHILL, Ph.D. Wayne State U. Coll. of Med., Dept. of Physiol. Chem., Detroit 7, Mich. *Research Assoc.* (5, 1946)

ORTEN, JAMES M., Ph.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Prof. of Physiol. Chem.* (2, 1936; 5, 1937)

ORTH, O. SIDNEY, Ph.D., M.D. U. of Wisconsin, Dept. of Anesthesiol., Madison 6. *Prof. and Chmn. of Dept.* (1, 1942; 3, 1944)

OSER, BERNARD L., Ph.D. Food and Drug Research Labs., Inc., Maurice Ave. at 58th St., Maspeth 78, New York City. *Pres. and Dir.* (5, 1945)

OSLER, ABRAHAM G., Ph.D. Johns Hopkins U., Dept. of Microbiol., Baltimore 5, Md. *Assoc. Prof.* (6, 1949)

OSTER, ROBERT H., Ph.D. U. of Maryland Sch. of Dent., Baltimore 1. *Prof. of Physiol.* (1, 1938)

OSTERBERG, ARNOLD E., Ph.D. Abbott Labs., Med. Dept., North Chicago, Ill. *Clin. Invest.* (2, 1933)

OSTERHOUT, MARIAN I., Ph.D. 450 East 63rd St., New York City. (1, 1927)

OSTERHOUT, W. J. V., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member Emer.* (1, 1910)

OTIS, ARTHUR B., Ph.D. U. of Florida Coll. of Med., Dept. of Physiol., Gainesville. *Prof. and Head of Dept.* (1, 1946)

OTT, WALTHER H., Ph.D. Merck Inst. for Therap. Research, Rahway, N. J. *Dir. of Animal Husbandry.* (5, 1949)

OUDIN, J., M.D. Institut Pasteur, 28 rue du Dr. Roux, Paris 15, France. *Head of Labs.* (6, 1954)

OVARY, ZOLTAN, M.D. Johns Hopkins U. Sch. of Med., Dept. of Microbiol., Baltimore 5, Md. *Research Assoc.* (6, 1957)

OVERMAN, JOHN R., M.D. Duke U. Sch. of Med., Dept. of Bacteriol., Durham, N. C. *Assoc. Prof.* (6, 1954)

OVERMAN, RICHARD R., Ph.D. U. of Tennessee Coll. of Med., Divs. of Physiol. and Med., Memphis. *Prof.; Dir., Clin. Physiol.* (1, 1946)

OWEN, CHARLES A., JR., M.D., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant in Clin. Pathol.* (1, 1953; 4, 1951)

OWEN, RAY D., Ph.D. California Inst. of Technology, Biology Div., Pasadena 4. *Prof.* (6, 1955)

OWEN, SEWARD E., Ph.D. 418 S. 20th Ave., Maywood, Ill. *Bacteriologist, VA Hosp., Hines, Ill.* (1, 1938)

OWENS, ALBERT H., JR., M.D. Johns Hopkins Sch. of Med., Baltimore 5, Md. *Instr. in Med.* (3, 1957)

PACE, DONALD M., Ph.D. U. of Nebraska, Dept. of Physiol., Lincoln. *Prof. and Chmn. of Dept.* (1, 1944)

PACE, NELLO, Ph.D. U. of California, Dept. of Physiol., Berkeley 4. *Prof.* (1, 1947)

PACK, GEORGE T., M.D. 139 E. 36th St., New York City 16. *Prof. of Clin. Surgery, N. Y. Med. Coll.; Attending Surgeon, Mem. Hosp.* (1, 1924)

PACKCHANIAN, ARDZROONY, Ph.D. U. of Texas Sch. of Med., Galveston. *Prof. of Microbiol.; Dir., Microbiol. Res. Lab.* (6, 1943)

PAGE, EDOUARD, Ph.D. U. of Montreal, Faculty of Sci., Dept. of Biol., Montreal, Canada. *Prof. and Head of Dept.* (1, 1947)

PAGE, ERNEST W., M.D. U. of California Hosp., Dept. of Obstetrics and Gynecol., San Francisco 22. *Prof.* (1, 1947)

PAGE, IRVINE H., M.D. Cleveland Clinic Fndn., Cleveland 6, Ohio. *Dir. of Research.* (1, 1937; 2, 1932)

PAGE, ROBERT G., M.D. U. of Chicago, Dept. of Med.,Chicago 37, Ill. *Assoc. Prof. and Asst. Dean.* (3, 1951)

PAINTER-MARCUS, ELIZABETH E., Ph.D. 438 Longcommon Rd., Riverside, Ill. (1, 1941)

PAIT, CHARLES F., M.D. Los Angeles City Health Dept., Los Angeles 12, Calif. *Dir. of Labs.* (6, 1953)

PALMER, JAMES K., Ph.D. 2820 Grove Ave., Richmond 21, Va. *Biochemist, Virginia Inst. for Sci. Research.* (2, 1957)

PALMER, JOHN W., Ph.D. Hyland Labs., 4501 Colorado Blvd., Los Angeles 39, Calif. (6, 1949)

PALMES, EDWARD D., Ph.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Indust. Med.* (1, 1958)

P'AN, S. Y., M.D. Pfizer Therap. Inst., Maywood, N. J. *Asst. Dir., Macrobiol. Research.* (3, 1941)

PANGBORN, MARY C., Ph.D. New York State Dept. of Health, Div. of Labs. and Research, Albany, N. Y. *Assoc. Biochemist.* (2, 1941)

PANOS, THEODORE C., M.D. U. of Arkansas Med. Ctr., Little Rock. *Prof. and Chmn., Pediatrics.* (4, 1956)

PAPAGEORGE, EVANGELINE, Ph.D. Emory U. Sch. of Med., Atlanta 22, Ga. (2, 1950)

PAPPENHEIMER, A. M., JR., Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Prof. of Biol.* (2, 1941; 6, 1938)

PAPPENHEIMER, JOHN R., Ph.D. Harvard Med. Sch., Boston, Mass. *Prof. of Physiol.; Career Invest., Amer. Heart Assn.* (1, 1946)

PAPPER, E. M., M.D. Presbyterian Hosp., 622 W. 168th St., New York City 32. *Prof. of Anesthesiol., Columbia U.* (3, 1949)

PARDEE, ARTHUR B., Ph.D. U. of California, Virus Lab., Berkeley 4. *Assoc. Prof.* (2, 1954)

PARDO, EFRAIN G., M.D. U. Nacional Autónoma de México, Escuela Nacional de Medicina, México, D. F. *Prof., Pharmacol.* (3, 1957)

PARFENTJEV, IVAN A., Sc.D. Jacob Ruppert Brewery, New York City 28. (6, 1955)

PARK, CHARLES R., M.D. Vanderbilt U. Sch. of Med., Dept. of Physiol., Nashville 5, Tenn. *Prof. and Head of Dept.* (1, 1955; 2, 1955)

PARK, EDWARDS A., M.D. Johns Hopkins Hosp., Baltimore, Md. *Prof. Emer. of Pediatrics, Johns Hopkins U.* (4, 1923)

PARK, JAMES T., Ph.D. Vanderbilt U. Sch. of Med., Dept. of Microbiol., Nashville 5, Tenn. (2, 1954)

PARK, JANE HARTING, Ph.D. Vanderbilt U. Med. Sch., Dept. of Physiol., Nashville 5, Tenn. *Asst. Prof.* (2, 1957)

PARKER, JOHN MULVIN, M.D. Charles E. Frosst & Co., Ltd., 350 Selby St., Montreal, Canada. *Pharmacologist.* (3, 1958)

PARKER, ROBERT F., M.D. Western Reserve U., Cleveland 6, Ohio. *Prof. of Microbiol.; Assoc. Prof. of Med.* (4, 1942; 6, 1935)

PARKINS, WILLIAM M., Ph.D. U. of Pennsylvania Sch. of Med., Harrison Dept. of Research Surgery, Philadelphia 4. *Assoc. Prof.* (1, 1939)

PARKS, ROBERT E., JR., M.D., Ph.D. U. of Wisconsin, Dept. of Pharmacol. and Toxicol., Madison 6. *Asst. Prof.* (3, 1956)

PARMER, LEO G., Ph.D., M.D. 61-34 188th St., Flushing 65, N. Y. (3, 1951)

PARPART, ARTHUR K., Ph.D. Princeton U., Guyot Hall, Princeton, N. J. *Prof. of Physiol.* (1, 1937)

PARR, LELAND W., Ph.D. George Washington U. Sch. of Med., Washington 5, D. C. *Prof. of Bacteriol.* (4, 1940)

PARRACK, HORACE O., Ph.D. Wright Air Develop. Ctr., Wright-Patterson AFB, Ohio. *Tech. Dir., R&D Prog.* (1, 1948)

PARRISH, D. B., Ph.D. Kansas State Coll., Dept. of Chem., Manhattan. *Assoc. Prof.* (5, 1951)

PARSONS, HELEN T., Ph.D. U. of Wisconsin, Madison 6. *Emer. Prof. of Home Econ.* (2R, 1929; 5R, 1933)

PARSONS, ROBERT J., M.D. Highland Alameda County Hosp., 2701 14th Ave., Oakland, Calif.

Patbologist; Dir. of Labs. (4, 1939)

PASCHKIS, KARL E., M.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Assoc. Prof. of Med. and Assoc. Prof. of Physiol.* (1, 1942)

PATERSON, JAMES C., M.D. U. of Western Ontario, Dept. of Med. Research, London, Canada. *Prof.* (4, 1957)

PATERSON, PHILIP Y., M.D. New York U. Coll. of Med., Dept. of Microbiol., New York City 16. (6, 1957)

PATRICK, HOMER, Ph.D. West Virginia U. Coll. of Agric., Poultry Dept., Morgantown. *Dept. Head.* (5, 1953)

PATT, HARVEY M., Ph.D. Argonne Natl. Lab., P.O. Box 299, Lemont, Ill. *Sr. Physiologist* (1, 1948)

PATTERSON, ELIZABETH K., Ph.D. Inst. for Cancer Research, Fox Chase, Philadelphia 11, Pa. *Assoc. Member.* (2, 1957)

PATTERSON, ERNEST L., Ph.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Research Chemist.* (2, 1956)

PATTERSON, JOHN L., JR., M.D. Med. Coll. of Virginia, Dept. of Med., Richmond 19. *Assoc. Prof. of Med.* (1, 1951)

PATTERSON, THOMAS L., Ph.D., Sc.D. California Inst. of Technology, Kerckhoff Marine Lab., Corona Del Mar. *Visting Res. Invest.* (1, 1920)

PATTERSON, WILBUR I., Ph.D. USDA, Eastern Utilization Res. and Develop. Div., Washington 25, D.C. *Chief, Dairy Products Lab.* (2, 1948)

PATTON, H. D., Ph.D., M.D. U. of Washington Sch. of Med., Dept. of Physiol. and Biophysics, Seattle 5. *Prof.* (1, 1947)

PATTON, MARY BROWN, Ph.D. Ohio State U., Sch. of Home Econ., Columbus 10. *Prof.* (5, 1953)

PAUL, JOHN R., M.D. Yale U. Sch. of Med., New Haven 11, Conn. *Prof. of Prev. Med.* (4R, 1927; 6, 1937)

PAUL, MARY F., Ph.D. Norwich Pharmacal Co., Norwich, N.Y. *Chief, Div. of Biol., Norwich Pharmacal Co., and Eaton Labs.* (2, 1953)

PAYNE, LOYAL COBB, D.V.M., Ph.D. U. of Nebraska Coll. of Agric., Lincoln 3. *Assoc. Prof. of Animal Patbol.* (1, 1958)

PAYNE, RICHARD W., M.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Assoc. Prof. of Pharmacol.* (3, 1954)

PAZUR, JOHN H., Ph.D. U. of Nebraska, Dept. of Biochem. and Nutrition, Lincoln 3. *Assoc. Prof.* (2, 1954)

PEACOCK, SAMUEL M., JR., M.D. Eastern Pennsylvania Psychiatric Inst., Philadelphia 29. *Sr. Med. Research Scientist.* (1, 1955)

PEARCE, JOHN MUSSER, M.D. 525 E. 68th St., New York City 21. *Prof. of Pathol., Cornell U. Med. Coll.; Attending Pathologist, N. Y. Hosp.* (4, 1942)

PEARCE, LOUISE, M.D. Trevenna Farm, Skillman, N. J. (4R, 1925)

PEARLMAN, WILLIAM H., Ph.D. Waldemar Med. Research Fndn., Port Washington, N.Y. *Assoc. Sci. Dir.* (2, 1946)

PEARSE, HERMAN E., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. of Surgery.* (4, 1932)

PEARSON, BJARNE, M.D. Detroit Inst. of Cancer Research, Detroit, Mich. *Res. Assoc.; Prof. Exptl. Pathol., Wayne State U.* (4, 1955)

PEARSON, HAROLD E., M.D., M.P.H. Los Angeles Country Hosp., Los Angeles, Calif. (6, 1948)

PEARSON, OLOF H., M.D. Sloan-Kettering Inst., New York City 21. *Assoc. Prof. of Med., Cornell U. Med. Coll.* (2, 1953)

PEARSON, P. B., Ph.D. Ford Fndn., Sci. and Engin. Program, 477 Madison Ave., New York City 22. (2, 1944; 5, 1940)

PEARSON, WILLIAM N., Ph.D. Vanderbilt U., Dept. of Biochem., Nashville, Tenn. *Asst. Prof.* (5, 1958)

PEASE, MARSHALL C., JR., M.D. Branchville Rd., R.F.D. 4, Ridgefield, Conn. (6R, 1920)

PECK, HAROLD M., M.D. Merck Inst. for Therapeutic Research, West Point, Pa. *Dir. of Patbol.* (3, 1953)

PECORA, LOUIS J., Ph.D. 1014 Broadway, Cincinnati 2, Ohio. *Chief, Physiol. Lab., PHS Occupational Health Prog.* (1, 1952)

PEISS, CLARENCE N., Ph.D. Loyola U. Stritch Sch. of Med., Dept. of Physiol., Chicago 12, Ill. *Assoc. Prof.* (1, 1952)

PELIKAN, EDWARD W., M.D. Boston U. Sch. of Med., Boston 18, Mass. *Assoc. Prof. of Pharmacol.* (3, 1952)

PENFIELD, WILDER G., M.D., D.Sc. McGill U., Montreal, Canada. *Dir., Montreal Neurol. Inst.* (1, 1932)

PENICK, GEORGE D., M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Assoc. Prof. of Patbol.* (4, 1955)

PENNEYS, RAYMOND, M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Instr. in Med.; Chief, Periph. Vasc. Sec., Philadelphia Gen. Hosp.* (1, 1951)

PENROD, KENNETH E., Ph.D. Duke U. Sch. of Med., Durham, N.C. *Prof. of Physiol.* (1, 1946)

PEOPLES, S. ANDERSON, M.D. U. of California, Sch. of Veterinary Med., Dept. of Pharmacol., Davis. *Prof. of Pharmacol. & Toxicol.* (3, 1937)

PERKINS, JOHN F., JR., M.D. U. of Chicago, Dept. of Physiol., Chicago 37, Ill. *Assoc. Prof.* (1, 1950)

PERKINSON, JESSE DEAN, JR., Ph.D. Atomic Energy Comm., Educ. and Training Br., DIA, Washington 25, D.C. *Chief.* (2, 1954)

PERL, EDWARD R., M.D. U. of Utah Coll. of Med., Dept. of Physiol., Salt Lake City 12. *Assoc. Prof.* (1, 1955)

PERLMAN, ELY, M.D. 118 Crescent Lane, Roslyn Heights, N.Y. *Res. Assoc., Mt. Sinai Hosp.; Chief, Allergy Clinic, L.I. Jewish Hosp.* (6, 1944)

PERLMAN, PRESTON L., Ph.D. Schering Corp., Bloomfield, N.J. *Dir., Biochem. Research.* (1, 1956; 3, 1956)

PERLMANN, GERTRUDE ERIKA, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (2, 1951)

PERLMUTT, JOSEPH H., Ph.D. U. of North Carolina Sch. of Med., Dept. of Physiol., Chapel Hill. *Assoc. Prof.* (1, 1955)

PERMAR, HOWARD H., M.D. Mercy Hosp., Pathologic Labs., Pittsburgh, Pa. *Pathologist.* (4, 1925)

PESKIN, JAMES C., Ph.D. U. of Rochester, Biol. Dept., Rochester, N.Y. *Assoc. Prof.* (1, 1955)

PETERMANN, MARY L., Ph.D. Sloan-Kettering Div. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Biochem.* (2, 1947)

PETERS, LAWRENCE, Ph.D., M.D. U. of Kansas Sch. of Med., Dept. of Pharmacol., Kansas City 12. *Prof. and Head of Dept.* (3, 1946)

PETERS, THEODORE, JR., Ph.D. Mary Imogene Bassett Hosp., Cooperstown, N.Y. *Res. Biochemist; Assoc. in Biochem., Columbia U.* (2, 1958)

PETERSEN, DONALD F., Ph.D. U. of California, Los Alamos Sci. Lab., P.O. Box 1663, Los Alamos, N. Mex. *Staff Member.* (3, 1956)

PETERSEN, WILLIAM E., Ph.D. U. of Minnesota, Div. of Dairy Husbandry, St. Paul 1. *Prof.* (1, 1947)

PETERSON, DANIEL W., Ph.D. U. of California, Dept. of Poultry Husbandry, Davis. *Asst. Prof.; Asst. Biochemist in Expt. Sta.* (5, 1957)

PETERSON, ELBERT A., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1954)

PETERSON, LYSLE H., M.D. U. of Pennsylvania Sch. of Med., Dept. of Physiol., Philadelphia 4. *Assoc. Prof., Physiol. & Surg. Physiol.* (1, 1950)

PETERSON, RUTH D., Ph.D. U. of Oregon Med. Sch., Biochem. Dept., Portland 1. *Asst. Prof.* (2, 1955)

PETERSON, WILLIAM H., Ph.D. U. of Wisconsin, Biochem. Bldg., Madison. *Prof. Emer. of Biochem.* (2R, 1919; 5R, 1936)

PETT, L. B., M.D., Ph.D. Dept. of Natl. Health and Welfare, Ottawa, Canada. *Chief, Nutrition Div.* (5, 1945)

PFAFFMANN, CARL, Ph.D. Brown U., Providence 12, R.I. *Prof. of Psychol.* (1, 1953)

PFANDER, WILLIAM H., Ph.D. U. of Missouri, 236 Mumford Hall, Columbia. *Prof. of Animal Husbandry.* (5, 1958)

PFEIFFER, CARL C., Ph.D., M.D. Emory U. Sch. of Med., Div. of Basic Health Sci., Atlanta 22, Ga. *Prof. of Pharmacol.; Dir. of Div.* (3, 1938)

PFIFFNER, JOSEPH J., Ph.D. Parke, Davis & Co., Research Labs., Detroit 32, Mich. *Biochemist (Research).* (1, 1931; 2, 1931; 5, 1946)

PHATAK, NILKANTH M., Ph.D. U. of Oregon Sch. of Dent., Dept. of Pharmacol., Portland. *Prof. and Head of Dept.* (3, 1941)

PHILIPS, FREDERICK S., Ph.D. Sloan-Kettering Inst. for Cancer Research, New York City 21. *Member.* (3, 1947)

PHILLIPS, HUGH JEFFERSON, Ph.D. Creighton U. Sch. of Med., Omaha, Nebr. *Asst. Prof. in Physiol.-Pharmacol.* (1, 1958)

PHILLIPS, PAUL H., Ph.D. U. of Wisconsin, Madison. *Prof. of Biochem.* (2, 1940; 5, 1938)

PHILLIPS, ROBERT ALLAN, M.D. U. S. Naval Med. Research Unit #2, APO 63, San Francisco, Calif. *Commanding Officer.* (1, 1938)

PHILPOT, VAN BUREN, JR., U. of Tennessee, Memphis. *Asst. Prof. of Pathol.* (4, 1957)

PICK, ERNST PETER, M.D. 19 E. 98th St., New York City 29. (3, 1940)

PICK, RUTH, M.D. Michael Reese Hosp., Med. Res. Inst., Chicago 16, Ill. *Asst. Dir., CV Dept.; Estab. Invest., Amer. Heart Assn.* (1, 1954)

PIEL, CAROLYN FORMAN, M.D. 2574 Green St., San Francisco 23, Calif. (4, 1950)

PIERCE, HAROLD B., Ph.D. U. of Vermont Coll. of Med., Dept. of Biochem., Burlington. *Prof. and Chmn. of Dept.* (2, 1929; 5, 1933)

PIERCE, HAROLD FISHER, Ph.D., M.D. 583 Park Rd., West Hartford 7, Conn. *Chief Med. Consultant, State Welfare Dept., Hartford.* (1, 1928)

PIERCE, IRA H., Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Emer. Prof. of Pharmacol.* (3R, 1933)

PIERCE, JOHN G., Ph.D. U. of California Med. Ctr., Dept. of Physiol. Chem., Los Angeles 24. *Assoc. Prof.* (2, 1954)

PIEZ, KARL ANTON, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Chemist.* (2, 1958)

PIGMAN, WARD, Ph.D. U. of Alabama, Biochem. Dept., Birmingham. *Assoc. Prof.; Co-Dir., Arthritis & Rheumatism Res. Lab.* (2, 1950)

PIKE, RUTH L., Ph.D. Pennsylvania State U., University Park. *Assoc. Prof. of Foods and Nutrition.* (5, 1957)

PILGERAM, L. O., Ph.D. St. Barnabas Hosp., Arteriosclerosis Res. Lab., Minneapolis, Minn. *Dir.; Asst. Prof., Physiol., U. of Minn.* (2, 1957)

PILIERO, SAM JOSEPH, Ph.D. New York Med. Coll., Flower-Fifth Ave. Hosps., New York City. *Assoc. Prof. of Anatomy.* (1, 1956)

PINCHOT, GIFFORD BRYCE, M.D. Johns Hopkins U., McCollum-Pratt Inst., Baltimore 18, Md. *Assoc. Prof.* (2, 1958)

PINCUS, GREGORY, Sc.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Research Dir.* (1, 1935)

PINCUS, I. J., M.D., D.Sc. U. of Pennsylvania Grad. Hosp., Philadelphia 46. *Asst. Prof., Grad. Sch. of Med.* (1, 1948)

PINKERTON, HENRY, M.D. St. Louis U. Sch. of Med., Dept. of Pathol., St. Louis, Mo. *Dir.* (4, 1931)

PINKSTON, JAMES O., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Assoc. Dean and Prof. of Physiol.* (1, 1936; 3, 1939)

PINSON, ERNEST A., Ph.D. 1109 Quincy St., S.E., Albuquerque, N. Mex. (1, 1943)

PIRANI, CONRAD L., M.D. U. of Illinois Coll. of Med., Dept. of Pathol., Chicago 12. *Prof.* (4, 1951)

PISANTY O., JOSE, M.D. U. of Nuevo Léon, Dept. of Physiol., Monterrey, N.L., Mexico. *Head of Dept.* (1, 1955)

PITTINGER, CHARLES B., M.D. State U. of Iowa, Iowa City. *Assoc. Prof. of Anesthesiol. and Pharmacol.* (3, 1954)

PITTMAN, MARGARET, Ph.D. Div. of Biologics Standards, NIH, Bethesda 14, Md. *Chief, Lab. of Bacterial Products.* (6, 1957)

PITTMAN, MARTHA S., Ph.D. 1909 Poyntz, Manhattan, Kans. (5R, 1933)

PITTS, GROVER C., Ph.D. U. of Virginia Sch. of Med., Dept. of Physiol., Charlottesville. *Assoc. Prof.* (1, 1951)

PITTS, ROBERT F., Ph.D., M.D. Cornell U. Med. Coll., Dept. of Physiol. and Biophysics., New York City 21. *Prof. and Head of Dept.* (1, 1934)

PLATNER, WESLEY S., Ph.D. U. of Missouri Sch. of Med., Columbia. *Prof. of Physiol.* (1, 1951)

PLAUT, GERHARD W. E., Ph.D. U. of Utah Sch. of

Med.. Salt Lake City 1. *Assoc. Res. Prof. of Biochem. and of Med.* (2, 1952)

PLESCIA, OTTO J., Ph.D. Rutgers, The State U., Inst. of Microbiol., New Brunswick, N. J. *Assoc. Prof. of Immunochem.* (6, 1956)

PLOTNIKOFF, NICHOLAS P., Ph.D. Merck Inst. for Therap. Res., West Point, Pa. *Res. Assoc., Pharmacodynamics.* (3, 1958)

PLUMMER, ALBERT J., M.D., Ph.D. Ciba Pharmaceut. Products, Inc., Summit, N. J. *Dir., Macrobiol.; Lect., Boston U.* (3, 1950)

POLGLASE, WILLIAM J., Ph.D. U. of British Columbia, Vancouver 8, Canada. *Assoc. Prof. of Biochem.* (2, 1957)

POLLACK, ABOU D. Baltimore City Hosps. Baltimore 24, Md. *Pathologist; Dept. of Pathol., Johns Hopkins U.* (4, 1953)

POLLACK, H., Ph.D., M.D. 70 E. 77th St., New York City 21. *Assoc. Physician, Mt. Sinai Hosp.; Assoc. Prof. Clin. Med., N. Y. U.* (1, 1933; 5, 1935)

POLLAK, OTAKAR J., M.D., Ph.D 9 Kings Highway, Dover, Del. *Patbologist.* (4, 1957)

POLLARD, MORRIS, D.V.M., Ph.D. U. of Texas Med. Br Galveston. *Prof. of Prev. Med.; Dir., Virus Research Lab.* (4, 1954)

POMERAT, CHARLES MARC, Ph.D. U. of Texas Med. Br., Galveston. *Prof. of Cytol.* (1, 1944)

POMMERENKE, W. T., Ph.D., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N. Y. *Assoc. Prof. of Obstetrics and Gynecol.* (1, 1947)

POND, SAMUEL E., Ph.D. U. of Hartford, Hillyer Coll., School of Arts & Sci., Hartford, Conn. *Assoc. Dean.* (1R, 1924)

PONDER, ERIC, M.D., Sc.D. Nassau Hosp., Mineola, N. Y. *Dir., Research Labs.* (1, 1931)

POPE, ALFRED, M.D. McLean Hosp., Waverley 79, Mass. *Neuropathologist; Assoc. Prof. of Neuropathol., Harvard Med. Sch.* (2, 1953)

POPENOE, EDWIN A., Ph.D. Brookhaven Natl. Lab., Med. Dept., Upton, N. Y. *Assoc. Biochemist.* (2, 1956)

POPPEN, JOHN R., M.D. 9000 Amestoy Ave., Northridge, Calif. *Capt. (MC) USN Ret.; Consultant.* (1, 1948)

POPPER, HANS, Ph.D., M.D. Mt. Sinai Hosp., Dept. of Pathol., New York City 29. *Dir. of Dept.* (4, 1942)

PORTER, CURT C., Ph.D. Merck Inst. for Therap. Research, West Point, Pa. *Research Assoc.* (2, 1954)

PORTER, EUGENE L., Ph.D. U. of Texas Med. Br., Galveston. *Prof. of Physiol.* (1, 1913)

PORTER, J. R., Ph.D. State U. of Iowa, Coll. of Med., Dept. of Bacteriol., Iowa City. *Prof. and Chmn. of Dept.* (6, 1950)

PORTER, JOHN C., Ph.D. U. of Texas Southwestern Med. Sch., Dept. of Physiol., Dallas 19. *Asst. Prof.* (1, 1958)

PORTER, JOHN W., Ph.D. VA Hosp., Radioisotope Serv., Madison, Wis. *Asst. Dir.; Assoc. Prof., U. of Wisconsin.* (2, 1953)

PORTER, ROBERT W., M.D., Ph.D. Walter Reed Army Inst. of Research, Washington 12, D. C. *Neurophysiologist.* (1, 1957)

PORTER, THELMA, Ph.D. Michigan State U., East Lansing. *Dean, Coll. of Home Econ.* (5, 1944)

POST, ROBERT LICKELY, M.D. 6310 Vanderbilt Med. Sch., Nashville 5, Tenn. *Asst. Prof. of Physiol.* (1, 1950)

POSTERNAK, JEAN M., M.D. U. of Geneva Med. Sch., Geneva, Switzerland. *Prof. of Physiol.* (1, 1951)

POTH, EDGAR J., M.D., Ph.D. U. of Texas Med. Br., Galveston. *Prof. of Surgery.* (1, 1946)

POTTER, RICHARD L., Ph.D. U. of Michigan, Radiation Lab., Ann Arbor. *Asst. Prof. of Biol. Chem.* (2, 1958)

POTTER, VAN R., Ph.D. U. of Wisconsin Med. Sch., McArdle Mem. Lab., Madison. *Prof. of Oncol.* (2, 1941)

POWELL, HORACE M., Sc.D. 5565 Washington Blvd., Indianapolis, Ind. *Research Advisor, Eli Lilly & Co.* (6, 1934)

POWER, MARSCHELLE H., Ph.D. Mayo Clinic, Rochester, Minn. *Prof. of Physiol. Chem., Mayo Fndn., U. of Minn.* (2, 1932)

PRADHAN, SACHINDRA N., M.D., D.T.M. Howard U. Coll. of Med., Washington 1, D. C. *Asst. Prof.* (3, 1958)

PRATT, ELMER B., M.D., Ph.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Med.* (1, 1958)

PRATT, FREDERICK H., M.D. 105 Hundreds Rd., Wellesley Hills, Mass. *Prof. Emer. of Physiol., Boston U. Sch. of Med.* (1R, 1919)

PREHN, RICHMOND T., M.D. U. of Washington Sch. of Med., Seattle. *Asst. Prof. of Pathol.* (4, 1956)

PREISLER, PAUL W., Ph.D., LL.B. Washington U. Sch. of Med., St. Louis 10, Mo. *Res. Asst. Prof.* (2, 1931)

PRESSMAN, DAVID, Ph.D. Roswell Park Mem. Inst., Buffalo 3, N. Y. *Dir. of Res., Biochem.; Res. Prof. Chem., U. of Buffalo.* (2, 1955; 6, 1949)

PRESTON, JAMES B., M.D. State U. of New York Coll. of Med., Dept. of Physiol., Syracuse 10. *Asst. Prof.* (3, 1956)

PRIBOR, HUGO C., M.D., Ph.D. Natl. Insts. of Health, Clin. Ctr., Bethesda 14, Md. *Resident in Pathol.* (4, 1957)

PRIBRAM, KARL H., M.D. Inst. of Living, Res. Labs., Hartford, Conn. *Dir. of Res. and Labs.; Lect., Yale U.* (1, 1951)

PRICE, HENRY L., M.D. U. of Pennsylvania, Dept. of Anesthesiol.. Philadelphia 4. *Assoc. Prof.* (1, 1955; 3, 1954)

PRICE, J. M., M.D., Ph.D. Cancer Research Hosp., Madison 6, Wis. *Assoc. Prof., Surgery, U. of Wisconsin Med. Sch.* (2, 1956)

PRINZMETAL, MYRON, M.D. 810 N. Rexford Dr., Beverly Hills, Calif. *Sr. Attending Phys., Cedars of Lebanon; Prof. Clin. Med., UCLA.* (3, 1941)

PRICE, VINCENT E., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Enzymes and Metab. Sec., Lab. of Biochem.* (2, 1953)

PROCITA, LEONARD, Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Asst. Prof. of Pharmacol.* (3, 1957)

PROCTOR, CHARLES D., Ph.D. Loyola U., Stritch Sch. of Med., Chicago 12, Ill. *Assoc. Prof. of Pharmacol. and Therap.* (3, 1957)

PROCTOR, DONALD F., M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Asst. Prof., Physiol. and Otolaryngol.* (1, 1955)

PROSSER, C. LADD, Ph.D. U. of Illinois, Dept. of Physiol., Urbana. *Prof.* (1, 1935)

PRUSOFF, WILLIAM H., Ph.D. Yale U. Sch. of Med., New Haven, Conn. *Asst. Prof. Pharmacol.* (2, 1956)

PUCK, THEODORE T., Ph.D. U. of Colorado Sch. of Med., Dept. of Biophysics, Denver 20. *Prof. and Head of Dept.* (6, 1955)

PUESTOW, CHARLES B., M.D., Ph.D. 25 E. Washington St., Chicago 2, Ill. *Clin. Prof. of Surgery, U. of Illinois Coll. of Med.* (1, 1934)

PUGSLEY, L. I., Ph.D. Dept. of Natl. Health and Welfare, Food and Drug Lab., Ottawa, Canada. *Asst. Dir., Sci. Serv.* (2, 1937)

PULLMAN, THEODORE N., M.D. U. of Chicago, Chicago 37, Ill. *Assoc. Prof. of Med.* (1, 1956)

PURPURA, DOMINICK P., M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof., Neurol. Surgery (Res.).* (1, 1957)

PUTMAN, EDISON W., Ph.D. U. of California, 337 Biochem.-Virus Lab., Berkeley 4. *Asst. Agric. Res. Biochemist.* (2, 1958)

PUTNAM, FRANK W., Ph.D. U. of Florida Coll. of Med., Dept. of Biochem., Gainesville. *Prof. and Chmn.* (2, 1947)

QUACKENBUSH, FORREST W., Ph.D. Purdue U., Dept. of Biochem., Lafayette, Ind. *Prof. and Head of Dept.* (2, 1946; 5, 1958)

QUAIFE, MARY L., Ph.D. Harvard U. Sch. of Public Health, Boston 15, Mass. *Assoc. in Nutrition.* (2, 1948; 5, 1951)

QUASTEL, J. H., D.Sc., Ph.D. McGill-Montreal Gen. Hosp. Res. Inst., Montreal, Canada. *Dir.; Prof. Biochem., McGill U.* (2, 1948)

QUEEN, FRANK B., M.D. U. of Oregon Sch. of Med., Portland. *Prof. of Pathol.* (4, 1941)

QUICK, ARMAND J., M.D., Ph.D. Marquette U. Sch. of Med., Dept. of Biochem., Milwaukee 3, Wis. *Prof. and Dir. of Dept.* (2, 1932; 3, 1937)

QUIGLEY, J. P., Ph.D. U. of Tennessee, Div. of Physiol., Memphis 3. *Prof. and Chief of Div.* (1, 1929; 3, 1945)

QUILLIGAN, J. J., JR., M.D. Coll. of Med. Evangelists, Los Angeles 33, Calif. *Prof. of Pediatrics; Dir., Virus Lab.* (6, 1950)

QUIMBY, FREEMAN HENRY, Ph.D. Office of Naval Research, Br. Office, 1000 Geary St., San Francisco 9, Calif. *Chief Scientist.* (1, 1950)

QUINN, EDMOND JOHN, Ph.D. 106 N. Lee Ave., Rockville Center, N.Y. (2R, 1927; 5R, 1933)

RAAB, WILHELM, M.D. U. of Vermont Med. Coll., Burlington. *Prof. of Exptl. Med.* (1, 1949)

RABEN, MAURICE S., M.D. Tufts U. Sch. of Med. Boston, Mass. *Asst. Prof. of Med.; Physician, New England Ctr. Hosp.* (1, 1955)

RABINOVITZ, MARCO, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1955)

RABINOWITZ, JESSE C., Ph.D. U. of California, Dept. of Biochem., Berkeley 4. *Assoc. Prof.* (2, 1954)

RABINOWITZ, JOSEPH L., Ph.D. Methodist Hosp., Texas Med. Ctr., Houston 25. *Assoc. Prof. of Biochem., Baylor U. Coll. of Med.* (2, 1957)

RABSON, ALAN S., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Pathologist.* (4, 1958)

RACHELE, JULIAN R., Ph.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Biochem.* (2, 1948)

RACKEMANN, FRANCIS MINOT, M.D. 263 Beacon St., Boston, Mass. *Bd. of Consultation, Massachusetts Gen. Hosp.* (6, 1923)

RACKER, EFRAIM, M.D. Public Health Research Inst., Div. of Nutrition and Physiol. New York City 9. *Chief, Div.* (2, 1948)

RADFORD, EDWARD P., JR., M.D. Haskell Lab., duPont Co., Newark, Del. *Physiologist; Lect. in Physiol., U. of Pa. Grad. Sch. Med.* (1, 1956)

RADIN, NORMAN S., Ph.D. Northwestern U. Med. Sch., Dept. of Biochem., Chicago 11, Ill. *Assoc. Prof.* (2, 1957)

RADOMSKI, JACK L., Ph.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Assoc. Prof. of Pharmacol.* (3, 1953)

RAFELSON, MAX E., JR., Ph.D. U. of Illinois Coll. of Med., Dept. of Biol. Chem., Chicago 12. *Assoc. Prof.* (2, 1955)

RAFFEL, SIDNEY, Sc.D., M.D. Stanford U., Dept. of Med. Microbiol., Stanford, Calif. *Exec. Head.* (6, 1938)

RAGAN, CHARLES, M.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Clin. Med.* (1, 1952)

RAHN, HERMANN, Ph.D. U. of Buffalo Sch. of Med. Buffalo, N.Y. *Prof. and Head of Physiol.* (1, 1944)

RAKESTRAW, NORRIS W., Ph.D. U. of California, Scripps Inst. of Oceanography, La Jolla. *Prof. of Chem.* (2, 1925)

RAKIETEN, NATHAN, Ph.D. South Shore Analytical and Research Lab., Box 47, Islip, N.Y. *Pharmacologist.* (1, 1941)

RALL, DAVID P., M.D., Ph.D. Natl. Cancer Inst., Clin. Pharmacol. and Exptl. Therap. Sec., NIH, Bethesda 14, Md. (3, 1956)

RALL, J. E., M.D., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Clin. Endocrinol. Br.* (1, 1955)

RALL, WILFRID, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Biophysicist.* (1, 1957)

RALLI, ELAINE P., M.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Med.* (1, 1934; 5, 1933)

RALSTON, H. J., Ph.D. Coll. of Physicians and Surgeons, 344-14th St., San Francisco 3, Calif. *Prof. of Physiol.* (1, 1947)

RAMEY, ESTELLE R., Ph.D. Georgetown U. Sch. of Med., Dept. of Physiol., Washington 7, D.C. *Asst. Prof.* (1, 1954)

RAMMELKAMP, CHARLES H., JR., M.D. Western Reserve U., Dept. of Med., Cleveland 6, Ohio. *Prof. of Med.; Assoc. Prof. Prev. Med.* (6, 1943)

RAMSEY, ROBERT WEBERG, Ph.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Physiol.* (1, 1939)

RANDALL, CHARLES C., M.D. U. of Mississippi Sch. of Med., Dept. of Microbiol., Jackson. *Prof. and Head of Dept.* (4, 1952; 6, 1958)

RANDALL, JAMES EDWIN, Ph.D. U. of Missouri Med. Ctr., Columbia. *Assoc. Prof. of Physiol. and Biophysics.* (1, 1956)

RANDALL, LOWELL O., Ph.D. D.Sc. Hoffmann-La Roche, Inc., Nutley 10, N.J. *Dir. of Pharmacol.*

(2, 1939; 3, 1950)

RANDALL, WALTER C., Ph.D. Loyola U. Stritch Sch. of Med., Dept. of Physiol., Chicago 12, Ill. *Chmn.* (1, 1943)

RANE, LEO, Ph.D. 571 Hammond St., Chestnut Hill 67, Mass. (6, 1942)

RANNEY, ROBERT E., Ph.D. G. D. Searle & Co., Div. of Biol. Research, Chicago 80, Ill. *Physiologist.* (1, 1955)

RANTZ, LOWELL A., M.D. Stanford U. Sch. of Med., San Francisco 15, Calif. *Prof. of Med.* (3, 1946)

RAPOPORT, S. Humboldt Universitat, Physiologisch-Chemisches Inst., Berlin N4, Germany. *Dir.* (2, 1941)

RAPPAPORT, IRVING, Ph.D. U. of California, Botany Dept., Los Angeles 24. *Asst. Res. Botanist.* (6, 1958)

RAPPORT, DAVID, M.D. Tufts U. Sch. of Med., Boston 11, Mass. *Prof. of Physiol.* (1, 1922)

RAPPORT, MAURICE M., Ph.D. Albert Einstein Coll. of Med., Dept. of Biochem., New York City 61. *Prof.* (2, 1950)

RASHKIND, WILLIAM J., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Cardiologist, Children's Hosp.* (1, 1952)

RASKA, SIGWIN B., Ph.D. M.D. Columbus Hosp., Chicago 14, Ill. (1, 1947)

RASMUSSEN, A. F., JR., Ph.D., M.D. U. of California Sch. of Med., Los Angeles 24. *Prof. Infectious Diseases (Virol.).* (6, 1949)

RATCLIFFE, HERBERT L., D.Sc. U. of Pennsylvania, Dept. of Pathol., Philadelphia 4. *Prof. of Comp. Pathol.; Pathologist, Zool. Soc.* (4, 1950)

RATHER, LELLAND J., M.D. Stanford U., Sch. of Med., Dept. of Pathol., San Francisco 15, Calif. *Prof.* (4, 1951)

RATNER, SARAH, Ph.D. Public Health Res. Inst. of City of N. Y., New York City 9. *Member, Div. Nutr. & Physiol.* (2, 1944)

RAU, G. CARL, Ph.D. U. of Kansas City Sch. of Pharmacy, Kansas City 10, Mo. (1, 1951)

RAULSTON, B. O., M.D. 538 So. Flower St., Los Angeles 17, Calif. *Prof. of Med. and Dean Emer., U. of Southern California.* (3R, 1942)

RAVDIN, I. S., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Barton Prof. of Surg.; Dir., Harrison Dept. Surg. Res.* (1, 1930, 4, 1930)

RAVEL, JOANNE M., Ph.D. U. of Texas, Clayton Fndn. Biochem. Inst., Austin. *Research Scientist.* (2, 1957)

RAWSON, RULON W., M.D. Sloan-Kettering Inst., New York City 21. *Member; Prof. of Med., Cornell U. Med. Coll.* (1, 1947)

RAYMOND, ALBERT L., Ph.D. G. D. Searle & Co., Chicago 80, Ill. *Vice Pres. in charge of Research and Develop.* (2, 1932)

RAYNAUD, MARCEL, M.D., Ph.D. Institut Pasteur, Blvd. Raymond Poincaré, Garches (S. & O.), France. *Chef de Service.* (6, 1957)

READ, WILLARD O., Ph.D. U. of South Dakota Sch. of Med., Vermillion. *Assoc. Prof. of Physiol.* (1, 1956)

REBACK, JOHN F., M.S., ScD. Sinai Hosp., Baltimore 5, Md. *Microbiologist.* (6, 1943)

REBUCK, JOHN W., M.D., Ph.D. Henry Ford Hosp., Dept. of Labs., Detroit 2, Mich. *Assoc. in Pathol.* (4, 1950)

REDFIELD, A. C., Ph.D. Woods Hole, Mass. *Prof. of Physiol. Emer., Harvard U.; Sr. Oceanographer Emer., Woods Hole Oceanographic Inst.* (1R, 1919)

REDISCH, WALTER, M.D. New York U., Res. Serv., Goldwater Mem. Hosp., Welfare Is., New York City 17. *Assoc. Prof., Clin. Med.* (1, 1956)

REED, CARLOS I., Ph.D. 1122 Northwood Dr., Guilford College Branch, Greensboro, N. C. *Prof. of Physiol. Emer., U. of Illinois.* (1R, 1923)

REED, EMERSON A., Ph.D. Hahnemann Med. Coll., Philadelphia, Pa. *Assoc. Prof. of Physiol.* (1, 1948)

REED, LESTER J., Ph.D. U. of Texas, Dept. of Chem., Austin 12. *Prof.* (2, 1952)

REESE, JOHN D., VA Hosp., Columbia, S. C. *Pathologist.* (4, 1949)

REEVE, E. B., B.M., B.Ch. U. of Colorado Sch. of Med., Denver 20. *Assoc. Prof. of Med.* (1, 1958)

REEVES, RICHARD E., Ph.D. Louisiana State U., Sch. of Med., New Orleans 12. *Assoc. Prof. of Biochem.* (2, 1956)

REHM, WARREN S., JR., Ph.D., M.D. U. of Louisville Sch. of Med., Louisville, Ky. *Prof. of Physiol.* (1, 1945)

REICH, HANS, Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Research Assoc.* (2, 1955)

REID, ALLEN FRANCIS, Ph.D. U. of Texas Southwestern Med. Sch., Dept. of Biophysics, Dallas 19. *Prof. and Chmn. of Dept.* (1, 1957)

REID, JAMES C., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1952)

REID, MARION ADELAIDE, Ph.D. Rutgers U., Douglass Coll., Dept. of Zool., New Brunswick, N. J. *Assoc. Prof.* (1, 1941)

REID, MARY E., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Biologist.* (5, 1947)

REILLY, JOSEPH, Ph.D. Cornell U., Med. Coll., New York City 21. *Asst. Prof. of Pharmacol.* (3, 1952)

REIMANN, HOBART A., M.D. Shiraz Med. Ctr., Shiraz, Iran. (4, 1933)

REIMANN, STANLEY P., M.D., ScD. 703 W. Phil-Ellena St., Philadelphia 19, Pa. *Emer. Dir., Inst. for Cancer Research.* (1, 1921; 4, 1924)

REINECKE, ROGER M., Ph.D., M.D. U. of Puerto Rico Sch. of Med., Dept. of Physiol., San Juan. *Prof. and Head of Dept.* (1, 1947)

REINFKE, E. P., Ph.D. Michigan State U., Dept. of Physiol. and Pharmacol., East Lansing. *Prof.* (1, 1950)

REINER, JOHN M., Ph.D. Simon Baruch Research Labs., Saratoga Spa., Saratoga Springs, N. Y. *Research Dir.* (1, 1947)

REINHARD, JOHN F., Ph.D. Wallace and Tiernan, Inc., Belleville, N. J. *Head, Pharmacol. Labs.* (3, 1952)

REINHARDT, WILLIAM O., M.D. U. of California Sch. of Med., Dept. of Anatomy, San Francisco 22. *Prof.* (1, 1950)

REINHOLD, JOHN G., Ph.D. U. of Pennsylvania Hosp., Philadelphia 4, Pa. *Prof. of Clin. Chem. in Med.* (2, 1936)

REISER, RAYMOND, Ph.D. Texas A & M Coll. System, Dept. of Biochem. and Nutr., College Station. *Prof.* (2, 1953)

REISNER, EDWARD H., M.D. 421 W. 113th St ., New York 25. *Asst. Attending Physician, St. Luke's Hosp.* (5, 1954)

REISSIG, MAGDALENA, M.D. Yale U. Sch. of Med., Sec. of Prev. Med., New Haven 11, Conn. *Research Assoc.* (6, 1957)

REISSMANN, KURT R., M.D. U. of Kansas Med. Sch., Kansas City 12. *Assoc. Prof. of Med.; Chief, Sec. of Exptl. Med.* (1, 1956)

REITHEL, FRANCIS J., Ph.D. U. of Oregon, Dept. of Chem., Eugene. *Prof.* (2, 1953)

REKERS, PAUL E., M.D. Cedars of Lebanon Hosp., Inst. for Med. Research, Los Angeles 27, Calif. *Research Assoc.* (4, 1950)

RELMAN, ARNOLD S., M.D. Boston U. Sch. of Med., Boston, Mass. *Assoc. Prof. of Med.* (1, 1957)

REMINGTON, JOHN W., Ph.D. Med. Coll. of Georgia, Dept. of Physiol., Augusta. *Prof.* (1, 1943)

REMINGTON, ROE E., Ph.D., D.Sc. 1227 Highland Ave., Hendersonville, N.C. (2R, 1930; 5R, 1934)

REMY, CHARLES N., Ph.D. VA Hosp., Med. Res. Div., Syracuse 10, N.Y. *Prin. Scientist; Instr. Biochem., State U. of N.Y.* (2, 1958)

RENFREW, ALICE G., Ph.D. U. of Pittsburgh, Mellon Inst., Pittsburgh, Pa. *Sr. Fellow, Fundamental Research.* (2, 1939)

RENKIN, EUGENE M., Ph.D. George Washington U. Sch. of Med., Washington 5, D.C. *Asst. Prof. of Physiol.* (1, 1954)

RENNICK, BARBARA R., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Asst. Prof. of Physiol.* (3, 1952)

RENOLD, ALBERT E., M.D. 185 Pilgrim Rd., Boston 15, Mass. *Assoc. in Med., Harvard Med. Sch.* (1, 1957)

RESSLER, CHARLOTTE, Ph.D. Cornell U. Med. Coll., New York City 21. *Asst. Prof. of Biochem.* (2, 1957)

REYNOLDS, MAY S., Ph.D. U. of Wisconsin Sch. of Home Econ., Dept. of Foods and Nutrition, Madison. *Prof.* (5, 1951)

REYNOLDS, MONICA, Ph.D. U. of Pennsylvania Sch. of Veterinary Med., Philadelphia 4. *Asst. Prof. of Physiol.* (1, 1954)

REYNOLDS, ORR E., Ph.D. Dept. of Defense, Washington 25, D.C. *Dir., Office of Sci.* (1, 1948)

REYNOLDS, SAMUEL R. M., Ph.D., ScD. U. of Illinois Coll. of Med., Dept. of Anatomy, Chicago 15. *Prof. and Head of Dept.* (1, 1932)

REZNIKOFF, PAUL, M.D. New York Hosp., 525 E. 68th St., New York City 21. *Prof. of Clin. Med., Cornell U., Med. Coll.* (1, 1927)

RHINES, RUTH, Ph.D., M.D. U. of Chicago, Dept. of Anatomy, Chicago 37, Ill. *Assoc. Prof.* (1, 1950)

RHOADS, C. P., M.D. Sloan-Kettering Inst., New York City 21. *Dir.; Sci. Dir. Mem. Ctr. for Cancer; Prof. Pathol., Cornell U.* (4, 1930)

RHOADS, JONATHAN E., M.D., D.Med.Sc. 131 W. Walnut La., Philadelphia 44, Pa. *Prof. of Surg., U. of Pa. Med. Sch.; Provost, U. of Pa.* (1, 1946)

RICE, CHRISTINE E., Ph.D. Animal Diseases Research Inst., Canada Dept. of Agric., Hull, Que., Canada. *Research Officer.* (6, 1938)

RICE, ELDON E., Ph.D. 12322 Elm St., Palos Park, Ill. *Research Chemist, Swift & Co.* (5, 1951)

RICE, HAROLD V., Ph.D. St. Paul's Hosp., Vancouver, Canada. (1, 1948)

RICE, JAMES C., Ph.D. U. of Mississippi Med. Ctr., Jackson. *Prof. of Pharmacol.; Chmn. of Dept.* (3, 1941)

RICH, ARNOLD RICE, M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Baxley Prof. Emer. of Pathol., Johns Hopkins U.* (4, 1924)

RICHARDS, ALFRED N., Ph.D., Sc.D. 737 Rugby Rd., Bryn Mawr, Pa. *Prof. Emer. of Pharmacol., U. of Pa.* (1R, 1900; 2R, 1906; 3R, 1909)

RICHARDS, DICKINSON W., M.D. Bellevue Hosp., First Med. Div., New York City 16. *Dir. & Physician; Lambert Prof. Med., Columbia U.* (1, 1953)

RICHARDS, FREDERIC M., Ph.D. Yale U. Sch. of Med., New Haven, Conn. *Asst. Prof. of Biochem.* (2, 1957)

RICHARDS, OSCAR W., Ph.D. American Optical Co., Research Ctr., Southbridge, Mass. *Research Supervisor for Biol.* (1, 1934)

RICHARDS, R. K., M.D. Abbott Labs., Res. Div., North Chicago, Ill. *Dir. Exptl. Therapy; Professorial Lect., Northwestern Med. Sch.* (1, 1938; 3, 1947)

RICHARDSON, ALFRED W., Ph.D. St. Louis U. Sch. of Med., St. Louis, Mo. *Assoc. Prof. of Physiol.* (1, 1952)

RICHARDSON, ARTHUR P., M.D. Emory U. Sch. of Med., Atlanta 22, Ga. *Dean; Prof. of Pharmacol.* (3, 1939)

RICHARDSON, JAMES A., Ph.D. Med. Coll. of South Carolina, Dept. of Pharmacol., Charleston 16. *Assoc. Prof.* (3, 1951)

RICHARDSON, LUTHER R., Ph.D. Texas A & M Coll., College Station. *Prof. of Biochem. and Nutrition.* (5, 1942)

RICHERT, DAN A., Ph.D. State U. of New York, Upstate Med. Ctr., Dept. of Biochem., Syracuse 10. *Assoc. Prof.* (2, 1951)

RICHTER, CURT P., Ph.D. Johns Hopkins Hosp., Phipps Psychiat. Clinic, Baltimore 5, Md. *Prof. Psycho-Biol., Johns Hopkins U.* (1, 1924)

RICHTER, GOETZ W., M.D. Cornell U. Med. Coll., Dept. of Pathol., New York City 21. *Assoc. Prof.* (4, 1954)

RICHTER, MAURICE N., M.D. 303 E. 20th St., New York City. *Prof. of Patbol., Bellevue Med. Ctr.; Dir., Dept. of Pathol., U. Hosp.* (4, 1931)

RICKETTS, HENRY T., M.D. U. of Chicago, Dept. of Med., Chicago, Ill. *Prof. of Med.* (1, 1940)

RIDDLE, OSCAR, Ph.D. Route 4, Plant City, Fla. (1R, 1919)

RIDER, J. ALFRED, M.D., Ph.D. U. of California Hosp., San Francisco. *Asst. Prof. of Med.* (3, 1957)

RIDLEY, ROGER W., M.D. Coll. of Med. Evangelists, Dept. of Anesthesiol., Loma Linda, Calif. *Asst. Prof.* (3, 1953)

RIEDERS, FREDRIC, Ph.D. Med. Examiner's Lab., Philadelphia 7, Pa. *Chief City Toxicologist; Assoc. Prof. Pharmacol., Jefferson Med. Coll.* (3, 1954)

RIEGEL, BYRON, Ph.D. G. D. Searle & Co., P.O. Box 5110, Chicago 80, Ill. *Dir. of Chem. Research.* (2, 1942)

RIEGEL, CECILIA, Ph.D. Lankenau Hosp., Biochem.

Lab., Philadelphia 31, Pa. *Biochemist.* (2, 1938)

RIESER, PETER, Ph.D. Marine Biological Lab., Woods Hole, Mass. (1, 1954)

RIGDON, R. H., M.D. U. of Texas Med. Br., Galveston. *Prof. of Patbol.* (4, 1941)

RIGGS, AUSTEN F., Ph.D. U. of Texas, Zool. Dept., Austin 12. *Asst. Prof.* (2, 1958)

RIGGS, DOUGLAS S., M.D. U. of Buffalo Sch. of Med., Dept. of Pharmacol., Buffalo 14, N.Y. *Prof. and Head of Dept.* (3, 1948)

RIGGS, LLOYD K., Ph.D. Box 101, Oakdale, N.Y. *Sr. Scientist, Evans Research and Develop. Corp.* (2R, 1929)

RIGGS, LORRIN A., Ph.D. Brown U., Providence 12, R.I. *Prof. of Psycbol.* (1, 1953)

RIGGS, THOMAS ROWLAND, Ph.D. U. of Michigan Med. Sch., Dept. of Biol. Chem., Ann Arbor. *Asst. Prof.* (2, 1954)

RIKER, WALTER F., M.D. Cornell U. Med. Coll., Dept. of Pharmacol., New York City 21. *Prof. and Cbmn.* (3, 1947)

RILEY, RICHARD F., Ph.D. U. of California Med. Ctr., Los Angeles 24. *Assoc. Prof. of Radiol. and Physiol. Chem.* (3, 1950)

RILEY, RICHARD L., M.D. 1901 Dixon Rd., Baltimore 9, Md. *Assoc. Prof. of Environ. Med. and of Med., Johns Hopkins U.* (1, 1948)

RING, GORDON C., Ph.D. U. of Miami Sch. of Med., Dept. of Physiol., Coral Gables 34, Fla. *Cbmn. of Dept.* (1, 1933)

RINZLER, SEYMOUR H., M.D. Beth Israel Hosp., 10 Livingstone Pl., New York City 3. (3, 1951)

RIOCH, DAVID M., M.D. Walter Reed Army Inst. of Res., Washington 12, D.C. *Dir., Neuropsycbiatry Div.* (1, 1931)

RITTENBERG, DAVID, Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Prof. and Cbmn. of Dept.* (2, 1939)

RIVERS, T. M., M.D., ScD. Rockefeller Inst. for Med. Research, New York City 21. *Member Emer.* (4, 1925; 6, 1921)

ROBB, JANE SANDS, ScD., M.D. Dartmouth Med. Sch., Hanover, N.H. *Visiting Prof. of Pbarmacol.* (1, 1924; 3, 1952)

ROBBINS, BENJAMIN H., M.D. Vanderbilt U. Sch. of Med., Nashville 4, Tenn. *Prof. of Anesthesiol.; Assoc. Prof. of Pharmacol.* (3, 1936)

ROBBINS, F. C., M.D. 2469 Wellington Rd., Cleveland Hts., Ohio. *Prof. Pediat., Western Reserve U.; Dir., Pediatrics, City Hosp.* (6, 1952)

ROBBINS, KENNETH C., Ph.D. Michael Reese Res. Fndn., 530 E. 31st St., Chicago 16, Ill. *Asst. Dir. of Research.* (6, 1950)

ROBBINS, MARY L., Ph.D. George Washington U. Sch. of Med. Washington 5, D.C. *Assoc. Prof. of Bacteriol.* (6, 1946)

ROBBINS, STANLEY L. Boston City Hosp., Mallory Inst. Pathol., Boston 18, Mass. *Assoc. Dir. & Assoc. Patbologist; Prof. Patbol., Boston U.* (4, 1952)

ROBERTS, EUGENE, Ph.D. City of Hope Med. Ctr., Dept. of Biochem., Duarte, Calif. *Cbmn. and Asst. Dir. of Research.* (2, 1950)

ROBERTS, JAY, Ph.D., Cornell U. Med. Coll., Dept. of Pharmacol., New York City 21. *Asst. Prof.* (3, 1956)

ROBERTS, JOSEPH T., M.D., Ph.D. VA Hosp., Buffalo, N.Y. *Chief of Cardiol. Sec.; Lect. in Med., U. of Buffalo Sch. of Med.* (1, 1947)

ROBERTS, KATHLEEN E., M.D. USPHS Hosp., 15th and Lake St., San Francisco, Calif. (1, 1956)

ROBERTS, LYDIA J., Ph.D. U. of Puerto Rico, Rio Piedras, P. R. *Prof. of Home Econ.* (5, 1933)

ROBERTS, SIDNEY, Ph.D. U. of California Sch. of Med., Los Angeles 24. *Prof. of Physiol. Chem.* (1, 1946; 2, 1950)

ROBERTSON, ELIZABETH CHANT, M.D., Ph.D. Hosp. for Sick Children, 555 University Ave., Toronto 2, Canada. *Assoc. Physician.* (5, 1939)

ROBERTSON, JAMES S., M.D., Ph.D. Brookhaven Natl. Lab., Med. Dept., Upton, N.Y. *Head, Med. Physics Div.* (1, 1954)

ROBERTSON, OSWALD H., M.D. Stanford U., Dept. of Biol., Palo Alto, Calif. *Prof. of Med.* (4R, 1932)

ROBERTSON, WILLIAM VAN B., Ph.D. U. of Vermont Coll. of Med., Dept. of Biochem., Burlington. *Prof.* (2, 1952)

ROBINS, ELI, M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Psychiatry.* (2, 1957)

ROBINSON, CHARLES SUMMERS, Ph.D. Vanderbilt U. Sch. of Med., Nashville 5, Tenn. *Prof. Emer. of Biochem.* (2R, 1925)

ROBINSON, ELLIOTT S., M.D., Ph.D. Elm St., R. F.D. 2, Lancaster, N.H. (6, 1935)

ROBINSON, G. CANBY, M.D. 320 Carpenter St., Greenport, N.Y. (1R, 1912)

ROBINSON, HARRY J., Ph.D., M.D. Merck Inst. for Therap. Research, Rahway, N.J. *Exec. Dir.* (3, 1946)

ROBINSON, HERBERT E., Ph.D. Swift & Co., Research Labs., Union Stock Yards, Chicago 9, Ill. *Dir. of Labs.* (5 , 1947)

ROBINSON, HOWARD W., Ph.D. Temple U. Med. Sch., Philadelphia 40, Pa. *Prof. Physiol. Chem.* (2, 1929)

ROBINSON, SID, Ph.D. Indiana U. Sch. of Med., Bloomington. *Prof. of Physiol.* (1, 1941)

ROBINSON, TRUE W., Ph.D. Med. Coll. of Alabama, Dept. of Physiol., Birmingham 3. (1, 1948)

ROBLIN, R. O., JR., Ph.D. Am. Cyanamid Co., 30 Rockefeller Plaza, New York City 20. *Gen. Mgr., Commercial Develop. Div.* (2, 1946)

ROBOZ, ELIZABETH, Ph.D. Stanford U., Sch. of Med., Lab. of Neurochem., Stanford, Calif. *Assoc. Prof.* (2, 1956)

ROBSCHEIT-ROBBINS, F. S., Ph.D. 169 Fishers Rd., Fishers, N.Y. (1, 1925; 4, 1930)

ROCHA E SILVA, MAURICIO, M.D. Instituto Biologico, Saõ Paulo, Brazil. *Chief, Biochem. and Pharmacol. Sec.* (3, 1952)

ROCKHOLD, WILLIAM T., Ph.D. Eaton Labs., Sec. of Pharmacol. 17 Eaton Ave., Norwich, N.Y. *Chief,* (3, 1958)

ROCKLAND, LOUIS B., Ph.D. 263 S. Chester Ave., Pasadena 5, Calif. *Chemist, USDA, Fruit and Vegetable Lab., ARS, WURDD.* (2, 1953)

ROCKSTEIN, MORRIS, Ph.D. New York U. Coll. of Med., Dept. of Physiol., New York City 16. *Assoc. Prof.* (1, 1955)

RODAHL, KAARE, M.D. Lankenau Hosp., Philadelphia 31, Pa. *Dir. of Research.* (1, 1952)

RODBARD, SIMON, Ph.D., M.D. U. of Buffalo Sch.

of Med., Chronic Disease Res. Inst., Buffalo 14, N.Y. *Dir., and Prof. Exptl. Med.* (1, 1942)

RODERUCK, CHARLOTTE E., Ph.D. Iowa State Coll., Dept. of Foods and Nutrition, Ames. *Prof.* (5, 1951)

RODKEY, FREDERICK L., Ph.D. Havard Med. Sch., Dept. of Biol. Chem., Boston 15, Mass. *Asst. Prof.* (2, 1955)

ROE, JOSEPH H., Ph.D. George Washington U. Sch. of Med., Washington 5, D. C. *Prof. of Biochem.; Exec. Officer of Dept.* (2, 1927; 5, 1933)

ROEDER, KENNETH D., D.Sc. Tufts U., Medford, Mass. *Prof. of Physiol.* (1, 1942)

ROEPKE, MARTIN H., Ph.D. USDA, ARS, Animal Dis. & Parasite Res. Div., Beltsville, Md. *Head, Chem. & Physics Sec.* (3, 1937)

ROEPKE, RAYMOND R., Ph.D. Amer. Cyanamid Co., Dept. of Pharmacol. Res., Pearl River, N. Y. *Group Leader.* (3, 1958)

ROGERS, FRED T., Ph.D., M.D. Dallas Med. and Surgical Clinic, 4105 Live Oak St., Dallas 1, Tex. (1R, 1917)

ROGERS, STANFIELD, M.D. U. of Tennessee Mem. Research Ctr., Knoxville. *Research Dir.* (4, 1952)

ROGOFF, JULIUS M., M.D., Sc. D. L. H. D. 45 S. Beach Dr., Belle Island, Rowayton, Conn. *Dir., Rogoff Fndn.* (1R, 1916; 3R, 1916)

ROHSE, WAYNE G., Ph.D. Loyola U., Stritch Sch. of Med., Chicago 12. Ill. *Asst. Prof. of Physiol.* (1, 1957)

ROINE, PAAVO K., Ph.D. Viikin Koetila, Malmi, Finland. *Prof. of Nutritional Chem., Helsinki U.* (5, 1950)

ROIZIN, LEON, M.D. New York State Psychiatric Inst., New York City 32. *Prin. Res. Scientist; Prof., Neuropathol., Columbia U.* (4, 1957)

ROLFE, DANIEL THOMAS, M.D. Meharry Med. Coll., Dept. of Physiol., Nashville 8, Tenn. *Dean; Prof. and Chmn. of Dept.* (1, 1950)

ROLL, PAUL M., Ph.D. Marquette U. Sch. of Med., Dept. of Biochem., Milwaukee 3, Wis. *Assoc. Prof.* (2, 1950)

ROMANOFF, ALEXIS L., Ph.D. Cornell U., Rice Hall, Ithaca, N. Y. *Prof. of Chem. and Embryol.* (1, 1953)

ROMANOFF, ELIJAH B., Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Research Scientist.* (1, 1957)

RONKIN, R. R., Ph.D. U. of Delaware, Newark. *Assoc. Prof., Dept. of Biol. Sci.* (1, 1956)

RONZONI, ETHEL, Ph.D. Washington U. Sch. of Med., Dept. of Psychiatry & Neurol., St. Louis 10, Mo. *Assoc. Prof. Biol. Chem.* (2, 1923)

ROOS, ALBERT, M.D. Washington U. Sch. of Med., Depts. of Physiol. and Surg., St. Louis 10, Mo. *Assoc. Prof. of Physiol.* (1, 1949)

ROOT, HOWARD F., M.D. Joslin Clinic, 15 Joslin Rd., Boston 15, Mass. *Lect. in Med., Havard Med. Sch.* (5, 1933)

ROOT, MARY AVERY, Ph.D. Eli Lilly & Co., Research Labs., Indianapolis 6, Ind. *Pharmacologist.* (3, 1952)

ROOT, RAYMOND W., Ph.D. City College of New York, New York City. *Prof. of Biol.; Head of Div. of Physiol.* (1, 1949)

ROOT, WALTER S., Ph.D. Columbia U. Coll. of P. & S., New York City 32. *Prof. of Physiol.* (1, 1932)

ROSAHN, PAUL D., M.D. 92 Grand St., New Britain, Conn. *Pathologist, New Britain Gen. Hosp.; Assoc. Clin Prof. Pathol., Yale U.* (4, 1934)

ROSE, CATHARINE S., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Assoc. in Pediatrics.* (2, 1953)

ROSE, HARRY M., M.D. Columbia U. Coll. of P. & S., Dept. of Microbiol., New York City 32. *Prof. and Chmn. of Dept.* (6, 1949)

ROSE, IRWIN A., Ph.D. Yale U. Sch. of Med., Dept. of Biochem., New Haven, Conn. *Asst. Prof.* (2, 1957)

ROSE, JERZY E., M.D. Johns Hopkins U. Sch. of Med., Dept. of Physiol., Baltimore 5, Md. *Assoc. Prof. of Physiol. and Psychiatry.* (1, 1949)

ROSE, JOHN C., M.D. Georgetown U. Sch. of Med., Dept. of Med., Washington 7, D. C. *Asst. Prof. of Med.* (1, 1958)

ROSE, NOEL R., Ph.D. U. of Buffalo Sch. of Med., Dept. of Bacteriol. and Immunol., Buffalo 14, N.Y. *Asst. Prof.* (6, 1956)

ROSE, WILLIAM C., Ph.D. U. of Illinois, Urbana. *Research Prof. of Biochem., Emer.* (2, 1912; 5, 1933)

ROSEBURY, THEODOR, D.D.S. Washington U. Sch. of Dent., St. Louis 10, Mo. *Prof. of Bacteriol.* (6, 1953)

ROSEMAN, SAUL, Ph.D. Rackham Arthritis Unit, U. Hosp., Ann Arbor, Mich. *Assoc. Prof. of Biol. Chem., U. of Mich.* (2, 1953)

ROSEN, FRED, Ph.D. Roswell Park Mem. Inst., Buffalo, N. Y. *Assoc. Cancer Research Scientist.* (5 1955)

ROSEN, HARRY, Ph.D. Wyeth Inst. for Med. Research, Box 8299, Philadelphia 1, Pa. *Research Scientist; Pharmacologist.* (3, 1957)

ROSENBERG, HANS REINHARD, D.Sc. E. I. du-Pont de Nemours and Co., Inc., Stine Lab., Newark, Del. *Mgr., Nutrition Sec.* (2, 1954; 5, 1958)

ROSENBLUETH, ARTURO, M.D. Instituto Nacional de Cardiologia, Ave. Cuauhtémoc 300, Mexico 7, D. F., Mexico. (1, 1932)

ROSENBLUM, IRA, Ph.D. Albany Med. Coll., Dept. of Pharmacol., Albany 8, N. Y. *Asst. Prof.* (3, 1956)

ROSENFELD, GEORGE, Ph.D. NAMRU #1, Berkeley, Calif. *Cdr., Med. Serv. Corps, USN (Allied Sci. Sec.); Res. Assoc., U. of California.* (1, 1957)

ROSENFELD, IRENE, Ph.D. U. of Wyoming, Dept. of Agric. Res. Chem., Laramie. *Res. Prof.* (2, 1958)

ROSENFELD, MORRIS, M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Assoc. Prof. of Pharmacol. and Exptl. Therap.* (3, 1934)

ROSENFELD, ROBERT S., Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc., Dept. of Steroid Biochem.* (2, 1956)

ROSENKRANTZ, HARRIS, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Research Biochemist; Head of Spectroscopy.* (2, 1957)

ROSENMAN, RAY H., M.D. 3969 Washington St., San Francisco 18, Calif. *Chief, Dept. Physiol., Brunn Inst., Mt. Zion Hosp.* (1, 1954)

ROSENOW, EDWARD C., M.D. Methodist Home, 3701

Bryant Ave. S., Minneapolis, Minn. (4R, 1913; 6R, 1915)

ROSENTHAL, HAROLD L., Ph.D. Rochester Gen. Hosp., Div. of Biochem., Rochester 8, N. Y. *Biochemist.* (2, 1957)

ROSENTHAL, OTTO, M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Assoc. Prof. of Biochem. in Surg., Harrison Dept. Surg. Res.* (2, 1946)

ROSENTHAL, S.R., M.D., Ph.D. U. of Illinois Coll. of Med., Chicago. *Assoc. Prof. Prev. Med., Dir., Inst. for Tuberculosis Res.* (1, 1948; 4, 1941)

ROSENTHAL, SANFORD M., M.D. Natl. Inst. of Arthritis & Metab. Diseases., NIH, Bethesda 14, Md. *Chief, Lab. Pharmacol. & Toxicol.* (3, 1925)

ROSS, BENJAMIN B., Ph.D. U. of Oregon Med. Sch., Dept. of Physiol., Portland 1. *Asst. Prof.* (1, 1958)

ROSS, JOSEPH F., M.D. U. of California Sch. of Med., Los Angeles 24. *Prof. of Med. and of Radiol. (Radiobiol); Assoc. Dean.* (4, 1941)

ROSS, OSCAR ALAN, M.D., Ph.D. Western Reserve U. Inst. of Pathol., Cleveland 6, Ohio. *Asst. Prof. of Patbol.* (4, 1957)

ROSS, RICHARD STARR, M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Med.* (1, 1957)

ROSSITER, R. J., M.D., Ph.D. U. of Western Ontario, London, Canada. *Prof. of Biochem.* (2, 1948)

ROSTORFER, HOWARD HAYES, Ph.D. Indiana U., Bloomington. *Prof. of Physiol.* (1, 1946)

ROTH, FRANKLIN ERNEST, Ph.D. Schering Corp., Bloomfield, N. J. *Sr. Pharmacologist.* (3, 1958)

ROTH, GEORGE B., M.D. 3814 T St. N. W., Washington 7, D. C. *Prof. Emer. of Pharmacol., George Washington U.* (1R, 1914; 3R, 1911)

ROTH, GRACE M., Ph.D. Mayo Clinic, Rochester, Minn. *Staff Consultant in Physiol.; Prof. of Physiol., Mayo Fndn.* (1, 1939)

ROTH, J. L. d'A., M.D., Ph.D. U. of Pa. Grad. Hosp., Philadelphia 46. *Dir., Gastroenterol. Res.; Assoc. Prof. of Gastroenterol. & Physiol.* (1, 1950)

ROTH, JAY S., Ph.D. Hahnemann Med. Coll., Philadelphia 2, Pa. *Assoc. Prof. of Biochem.* (2, 1956)

ROTH, L. W., Ph.D. Riker Labs., Inc., Northridge, Calif. (1, 1947; 3, 1950)

ROTH, LLOYD J., Ph.D., M.D. U. of Chicago, Dept. of Pharmacol., Chicago, Ill. *Chmn. of Dept.* (3, 1955)

ROTHCHILD, IRVING, Ph.D., M.D. Western Reserve U., Dept. of Obstetrics and Gynecol., Cleveland 6, Ohio. (1, 1949)

ROTHEMUND, PAUL W. K., Dr.-Ing. Box 64, Yellow Springs, Ohio. *Consultant Chemist and Chem. Engineer.* (2, 1940)

ROTHLIN, ERNST, M.D. Sonnenweg 6, Basle, Switzerland. *Prof. of Med.* (3, 1951)

ROTHSTEIN, A., Ph.D. U. of Rochester Sch. of Med., Rochester 20, N. Y. *Assoc. Prof. Pharmacol.; Chief, Physiol. Sec., AEC Proj.* (1, 1949; 3, 1952)

ROTHSTEIN, MORTON, Ph.D. U. of California Sch. of Med., Dept. of Biochem., San Francisco 22. *Assoc. Res. Biochemist.* (2, 1956)

ROUGHTON, FRANCIS JOHN WORSLEY, Ph.D. U. of Cambridge, Cambridge, England. *John Humphrey Plummer Prof. of Colloid Sci.* (1H, 1957)

ROUS, PEYTON, M.D., Sc.D. Rockefeller Inst. for Med. Research, New York City 21. *Member Emer.* (4, 1913)

ROUSER, GEORGE, Ph.D. City of Hope Med. Ctr., Biochem. Dept., Duarte, Calif. *Res. Assoc.* (2, 1958)

ROUTH, JOSEPH I., Ph.D. State U. of Iowa Med. Research Ctr., Dept. of Biochem., Iowa City. *Prof* (2, 1942)

ROVENSTINE, EMERY ANDREW, M.D. New York U.-Bellevue Med. Ctr., Dept. of Anesthesiol., New York City 16. *Prof.* (3, 1944)

ROWLEY, DONALD ADAMS, M.D. U. of Chicago, Dept. of Pathol., Chicago 37, Ill. *Asst. Prof.* (4, 1956)

ROWNTREE, JENNIE I., Ph.D. 6318-21st St., N. E., Seattle, Wash. (5, 1933)

ROWNTREE, L. G., M.D., Sc.D. duPont Bldg., E. Flagler St. at Second Ave., Miami, Fla. (1R, 1911; 2R, 1910; 3R, 1908)

RUBENSTEIN, BORIS B., Ph.D., M.D. 1357 E. Madison Park Ave., Chicago 15, Ill. (1, 1934)

RUBIN, BERNARD, Ph.D. Squibb Inst. for Med. Research, New Brunswick, N. J. *Research Pharmacologist.* (3, 1954)

RUBIN, SAUL H., Ph.D. Hoffmann-La Roche, Inc., Nutley 10, N. J. *Dir., Applied Research.* (2, 1947; 5, 1947)

RUCH, THEODORE C., Ph.D. U. of Washington, Dept. of Physiol. & Biophysics, G405 Health Sci. Bldg., Seattle 5. *Prof. and Exec. Officer.* (1, 1933)

RUCHMAN, ISAAC, Ph.D. Wm. S. Merrell Co., Cincinnati 15, Ohio. *Head of Microbiol.* (6, 1948)

RUDIN, DONALD O., M.D. Eastern Pennsylvania Psychiatric Inst., Philadelphia. *Dir. of Basic Research.* (1, 1955)

RUDNEY, HARRY, Ph.D. Western Reserve U. Sch. of Med., Dept. of Biochem., Cleveland 6, Ohio. *Assoc. Prof.* (2, 1955)

RUDOLPH, GUILFORD G., Ph.D. U. of Maryland Sch. of Med., Baltimore 1. *Assoc. Prof. of Biol. Chem.* (1, 1957)

RUEGAMER, W. R., Ph.D. VA Hosp., Radioisotope Unit, Syracuse 10, N. Y. *Prin. Scientist; Asst. Prof. of Biochem., State U. of N. Y.* (2, 1955)

RUHE, C. H. WILLIAM, M.D. U. of Pittsburgh, Sch. of Med., Pittsburgh 13, Pa. *Assoc. Prof. of Physiol. and Pharmacol.* (1, 1951)

RUSCH, HAROLD PAUL, M.D. U. of Wisconsin Med. Sch., McArdle Mem. Lab., Madison 6. *Prof. of Oncol.; Dir. of Lab.* (4, 1940)

RUSHMER, ROBERT F., M.D. U. of Washington Sch. of Med., Dept. of Physiol. and Biophysics, Seattle 5. *Prof.* (1, 1949)

RUSKIN, ARTHUR, M.D. U. of Texas Med. Br., Galveston. *Assoc. Prof. of Int. Med.* (1, 1957)

RUSOFF, LOUIS L., Ph.D. Louisiana State U., Baton Rouge. *Nutritionist.* (5, 1948)

RUSSELL, JANE A., Ph.D. Emory U., Dept. of Biochem., Atlanta 22, Ga. *Assoc. Prof.* (1, 1939)

RUSSELL, WILLIAM O., M.D. U. of Texas Med. Ctr., M. D. Anderson Hosp., Houston. *Pathologist-in-Chief.* (4, 1955)

RUSSFIELD, AGNES BURT, M.D. Massachusetts Gen Hosp., Boston 14. *Asst. Pathologist; Assoc. in Pathol., Havard Med. Sch.* (4, 1956)

RUST, JOHN HOWARD, D.V.M., Ph.D. Armed Forces

Inst. of Pathol., WRAMC, Washington 25, D. C. (4, 1957)

RUSTIGIAN, ROBERT, Ph.D. Tufts U. Sch. of Med., Dept. of Bacteriol. and Pathol., Boston, Mass. *Asst. Prof.* (6, 1951)

RUTMAN, ROBERT J., Ph.D. U. of Pennsylvania, Dept. of Chem., Philadelphia 4. *Research Assoc.* (2, 1957)

RUTTER, WILLIAM J., Ph.D. U. of Illinois, Dept. of Chem., Urbana. *Asst. Prof. of Biochem.* (2, 1958)

RYAN, ANDREW HOWARD, M.D. Chicago Med. Sch., Chicago 12, Ill. *Assoc. Prof. of Physiol. and Pharmacol.* (1, 1912)

RYTAND, DAVID A., M.D. Stanford U., Sch. of Med., San Francisco 15, Calif. *Prof. of Med.* (3, 1946)

SABIN, ALBERT B., M.D. Children's Hosp. Research Fndn., Cincinnati, Ohio. *Prof. of Research Pediatrics, U. of Cincinnati.* (6, 1946)

SABLE, HENRY Z., M.D., Ph.D. Western Reserve U., Dept. of Biochem., Cleveland 6, Ohio. *Assoc. Prof.* (2, 1954)

SACHS, ERNEST, M.D. 28 Marlborough Rd., North Haven, Conn. *Instr. in Surgery, Yale U.; Prof. Emer., Clin. Neurol. Surg., Wash. U.* (1R, 1910)

SACHS, HOWARD, Ph.D. Western Reserve U., Sch. of Med., Dept. of Physiol., Cleveland 6, Ohio. *Instr.* (2, 1957)

SACKS, JACOB, Ph.D., M.D. U. of Arkansas, Dept. of Chem., Fayetteville. *Prof.* (1, 1948; 3, 1933)

SACKTOR, BERTRAM, Ph.D. Chem. Warfare Labs., Directorate of Med. Research, Army Chemical Center, Md. *Biochemist.* (2, 1957)

SAH, PETER P.T., Ph.D. U. of California Sch. of Veterinary Med., Dept. of Pharmacol., Davis. *Prof.* (3, 1941)

SAHYUN, MELVILLE, Ph.D. Sahyun Research Lab., 316 Castillo St., Santa Barbara, Calif. *Dir. and Pres.* (2, 1932)

SAKAMI, WARWICK, Ph.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof. of Biochem.* (2, 1949)

SALERNO, PAUL R., M.D., Ph.D. Western Reserve U., Dept. of Pharmacol., Cleveland 6, Ohio. *Asst Prof.* (3, 1955)

SALISBURY, PETER F., Ph.D., M.D. Cedars of Lebanon Hosp., Los Angeles 29, Calif. *Consultant.* (1, 1957)

SALK, JONAS E., M.D. U. of Pittsburgh Sch. of Med., Virus Research Lab., Pittsburgh, Pa. *Commonwealth Prof. of Exptl. Med.* (6, 1947)

SALLACH, H. J., Ph.D. U. of Wisconsin, Service Mem. Inst., Dept. of Physiol. Chem., Madison 6. *Asst. Prof.* (2, 1957)

SALMON, W. D., A.M. Alabama Polytechnic Inst., Auburn. *Animal Nutritionist and Prof.* (2, 1929; 5, 1933)

SALOMON, KURT, M.D., Ph.D. U. of Rochester, Rochester 20, N. Y. *Assoc. Prof. Radiation Biol. & Biochem.; Chief, Radiation Chem. Sec., AEP.* (2, 1952)

SALTMAN, PAUL, Ph.D. U. of Southern California, Dept. of Biochem., Los Angeles 7. *Assoc. Prof.* (2, 1957)

SALVIN, SAMUEL B., Ph.D. U. S. Public Health Serv., Rocky Mountain Lab., Hamilton, Mont. *Scientist Dir.* (6, 1952)

SAMARAS, STELIOS C., M.D. Creighton U., Sch. of Med., Omaha, Nebr. *Assoc. Prof. of Pathol.* (1, 1955)

SAMET, PHILIP, M.D. Mt. Sinai Hosp., Miami Beach 40, Fla. *Dir., Cardio-pulmonary Lab.; Assoc. Prof. of Physiol., U. of Miami.* (1, 1956)

SAMPSON, JOHN J., M.D. 2211 Post St., San Francisco, Calif. *Clin. Prof. of Med., U. of California.* (1, 1932)

SAMPSON, MYRA M., Ph.D. Smith Coll., Northampton, Mass. *Prof. Emer. of Zool.* (5, 1935)

SAMSON, FREDERICK EUGENE, JR., Ph.D. U. of Kansas Sch. of Med., Dept. of Physiol., Lawrence. *Assoc. Prof.* (1, 1957)

SAMUELS, LEO T., Ph.D. U. of Utah Coll. of Med., Dept. of Biol. Chem., Salt Lake City 1. *Prof. and Head of Dept.* (2, 1941; 3, 1937; 5, 1949)

SANADI, D. RAO, Ph.D. U. of California, Dept. of Physiol. Chem., Berkeley 4. *Asst. Prof.* (2, 1952)

SANDELS, MARGARET R., Ph.D. Florida State U., Tallahassee. *Dean Emer., Sch. of Home Econ.; Prof. of Nutrition.* (5, 1933)

SANDIFORD, IRENE, Ph.D. c/o Dr. James H. Saint, 2435 State St., Santa Barbara, Calif. (2R, 1925; 5R, 1933)

SANDOW, ALEXANDER, Ph.D. New York U., Washington Square Coll. of Arts and Sci., New York City. *Prof. of Biol.* (1, 1945)

SANDWEISS, D. J., M.D. 15201 W. McNichols Rd., Detroit 35, Mich. *Asst. Prof., Clin. Med., Wayne State U.; Attending Phys., Med., Harper Hosp.* (1, 1944)

SANFORD, ARTHUR H., M.D. Mayo Clinic, Div. of Clin. Labs., Rochester, Minn. *Emer. Head.* (6R, 1920)

SANFORD, KATHERINE KOONTZ, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biologist (Cytol.).* (4, 1957)

SAN PIETRO, ANTHONY, Ph.D. Johns Hopkins U., McCollum-Pratt Inst., Baltimore 18, Md. *Asst. Prof. of Biol.* (2, 1957)

SANTOS, FRANCISCO O., Ph.D. U. of the Philippines Coll. of Agric., Laguna. *Asst. Dean; Prof. of Agric. Chem.* (5, 1936)

SAPHIR, OTTO, M.D. Michael Reese Hosp., Chicago 16, Ill. *Dir. of Patbol.; Clin. Prof. of Patbol., U. of Illinois.* (4, 1927)

SAPIRSTEIN, LEO ABRAHAM, Ph.D., M.D. Ohio State U., Dept. of Physiol., Columbus 10. *Prof.* (1, 1950)

SARETT, HERBERT P., Ph.D. Mead Johnson & Co., Res. Labs., Evansville 21, Ind. *Dir., Nutritional and Biochem. Res.* (2, 1946; 5, 1947)

SARGENT, FREDERICK, II, M.D. U. of Illinois, 401 Natural History Bldg., Urbana. *Assoc. Prof. of Physiol.* (1, 1952)

SARMA, PADUBIDRI S., Ph.D. U. of Madras, Madras, India. *Prof. of Biochem.* (5, 1958)

SARNOFF, STANLEY J., M.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Cardiovascular Physiol.* (1, 1949)

SARTORIUS, OTTO W., M.D. Syracuse U. Coll. of Med., Syracuse, N. Y. *Asst. Prof. of Physiol.* (1, 1950)

SASLAW, SAMUEL, Ph.D., M.D. Ohio State U., Columbus 10. *Assoc. Prof. in Med. and Bacteriol.*

(6, 1950)

SATTERFIELD, G. HOWARD, A.M. U. of North Carolina State Coll. of Agric. and Engin., Raleigh. *Prof. of Biochem.* (2, 1944; 5, 1941)

SAUBERLICH, H. E., Ph.D. Alabama Polytech. Inst., Dept. Animal Husb. & Nutr., Auburn. *Prof.; Animal Nutritionist.* (2, 1951; 5, 1952)

SAUL, LEON JOSEPH, M.D. 111 No. 49th St., Philadelphia 39, Pa. *Prof. of Clin. Psychiatry, U. of Pennsylvania Sch. of Med.* (1, 1933)

SAUNDERS, FRANCIS J., Ph.D. G. D. Searle & Co., Chicago, Ill. *Chief Endocrinologist, Div. of Biol. Research.* (1, 1956)

SAUNDERS, J. PALMER, Ph.D. Div. of Research Grants, NIH, Bethesda 14, Md. *Pharmacologist.* (3, 1956)

SAUNDERS, PAUL R., Ph.D. U. of Southern California Sch. of Med., Dept. of Pharmacol., Los Angeles 7. *Assoc. Prof.* (3, 1958)

SAVARD, F. G. KENNETH, D.Sc. U. of Miami Sch. of Med., Miami 36, Fla. *Assoc. Prof. of Biochem.; Chief, Endocrine Lab., Dept. of Med.* (2, 1953)

SAWYER, CHARLES H., Ph.D. U. of California Sch. of Med., Dept. of Anatomy, Los Angeles 24. *Prof. and Chmn. of Dept.* (1, 1949)

SAWYER, MARGARET E. MacKAY, Ph.D. Queen's U., Dept. of Physiol., Kingston, Canada. (1, 1935)

SAWYER, WILBUR H., Ph.D., M.D. Columbia U., Coll. of P. & S., Dept. of Pharmacol., New York City 32. *Assoc. Prof.* (1, 1953; 3, 1958)

SAYERS, GEORGE, Ph.D. Western Reserve U. Sch. of Med., Dept. of Physiol., Cleveland 6, Ohio. *Prof.* (1, 1948; 3, 1947)

SAZ, HOWARD J., Ph.D. Louisiana State U. Sch. of Med., New Orleans 12. *Assoc. Prof. of Pharmacol.* (2, 1957)

SCANTLEBURY, RONALD E., Ph.D. Div. of Research Grants, NIH, Bethesda 14, Md. *Chief, Research Fellowships Br.* (1, 1948)

SCARPELLI, DANTE G., M.D. Ohio State U. Coll. of Med., Columbus. *NIH Sr. Research Fellow in Pathol.* (4, 1957)

SCHABEL, F. M., JR., Ph.D. Southern Research Inst., Birmingham 5, Ala. *Head, Chemotherapy Div.; Microbiologist, Baptist Hosps.* (4, 1956)

SCHACHMAN, HOWARD K., Ph.D. U. of Calif., Biochem. and Virus Lab., Berkeley 4. *Assoc. Prof. of Biochem.; Assoc. Res. Biochemist.* (2, 1955)

SCHAEFER, ARNOLD E., Ph.D. Natl. Insts. of Health, Interdepartmental Committee on Nutrition, Bethesda 14, Md. (5, 1950)

SCHAEFER, KARL ERNST, M.D. U. S. Naval Med. Res. Lab., New London, Conn. *Physiol. Supervisor; Head, Physiol. Br.* (1, 1953)

SCHAFFER, NORWOOD K., Ph.D., M.D. Chem. Warfare Labs., Enzyme Chem. Br., Army Chemical Ctr., Md. *Biochemist.* (2, 1954)

SCHALES, OTTO, D.Sc. 3103 State St. Dr., New Orleans, La. *Dir. Chem. Res., Ochsner Fndn.; Prof. Biochem., Tulane U.* (2, 1944)

SCHALLEK, WILLIAM B., Ph.D. Hoffmann-La Roche, Inc., Nutley 10, N. J. *Sr. Physiologist.* (1, 1951; 3, 1957)

SCHANKER, LEWIS S., Ph.D. Natl. Heart Inst., Lab. of Chem. Pharmacol., NIH, Bethesda 14, Md. *Pharmacologist.* (3, 1958)

SCHARLES, FREDERICK H., M.D. 333 N. Highland Ave., Los Angeles 36, Calif. (5, 1935)

SCHATTENBERG, HERBERT JOHN, M.D. Lab. of Clin. Pathol., 222 Med. Arts Bldg., San Antonio, Tex. *Dir.* (4, 1940)

SCHAYER, RICHARD W., Ph.D. Merck Inst. for Therap. Research, Rahway, N. J. *Research Assoc.* (1, 1957; 2, 1952)

SCHEER, BRADLEY T., Ph.D. U. of Oregon, Eugene. *Prof. of Biol.* (1, 1952)

SCHEIN, ARNOLD H., Ph.D. U. of Vermont Sch. of Med., Dept. of Biochem., Burlington. *Assoc. Prof.* (2, 1958)

SCHENCK, JAY R., Ph.D. Abbott Labs., North Chicago, Ill. *Research Biochemist.* (2, 1952)

SCHENKEN, JOHN R., M.D. U. of Nebraska Coll. of Med., Dept. of Pathol., Omaha. *Prof. and Chmn.* (4, 1942)

SCHEPERS, GERRIT W. H., M.D., D.Sc. Haskell Lab., du Pont de Nemours & Co., Newark, Del. *Patbologist.* (4, 1957)

SCHER, ALLEN M., Ph.D. U. of Washington, Dept. of Physiol. and Biophysics, Seattle 5. *Assoc. Prof.* (1, 1955)

SCHERAGA, HAROLD A., Ph.D. Cornell U., Dept. of Chem., Ithaca, N. Y. *Prof.* (2, 1955)

SCHERAGO, MORRIS, D.V.M. U. of Kentucky, Dept. of Bacteriol., Lexington. *Prof. and Head of Dept.* (6, 1948)

SCHERER, WILLIAM F., M.D. U. of Minnesota Med. Sch., Minneapolis 14. *Assoc. Prof. of Bacteriol. and Immunol.* (6, 1954)

SCHERP, HENRY W., Ph.D. Natl. Inst. of Dental Research, NIH, Bethesda 14, Md. *Chief, Lab. of Microbiol.* (6, 1940)

SCHICK, BELA, M.D. 1045 Park Ave., New York City 28. *Dir. Pediatrics Dept., Beth El Hosp., Brooklyn.* (6, 1924)

SCHIFFRIN, MILTON J., Ph.D. Hoffmann-La Roche, Inc., Chicago, Ill. *Asst. Dir., Dept. of Clin. Research.* (1, 1943)

SCHILLER, JOSEPH, M.D., Ph.D. VA Hosp., 408 First Ave., New York City. *Staff.* (1, 1949)

SCHILLER, SARA, Ph.D. LaRabida-U. of Chicago Inst., Chicago 49, Ill. *Research Assoc. (Asst. Prof.) U. of Chicago.* (2, 1955)

SCHILLING, JOHN A., M.D. U. of Oklahoma Med. Ctr., Oklahoma City 4. *Prof. of Surgery.* (4, 1956)

SCHLAMOWITZ, MAX, Ph.D. Roswell Park Mem. Inst., Div. of Biochem., Buffalo 3, N. Y. (2, 1953)

SCHLEGEL, J. U., M.D., Ph.D. Strong Mem. Hosp., Rochester 20, N. Y. *Asst. Prof. of Urological Surg.; Dir., Urological Res. Lab.* (1, 1951)

SCHLENK, FRITZ, Ph.D. Argonne Natl. Lab., Div. of Biol. and Med. Research, Lemont, Ill. *Sr. Biochemist.* (2, 1942)

SCHLESINGER, R., WALTER, M.D. St. Louis U. Sch. of Med., Dept. of Microbiol., St. Louis 4, Mo. *Prof. and Dir. of Dept.* (6, 1956)

SCHLOMOVITZ, BENJAMIN H., M.D. 1210 Majestic Bldg., 231 W. Wisconsin Ave., Milwaukee, Wis. (1, 1919)

SCHLUMBERGER, HANS G., M.D. U. of Arkansas Med. Ctr., Little Rock. *Prof. of Patbol.* (4, 1945)

SCHMEISSER, HARRY C., Ph.D., M.D. U. of Ten-

nessee, Memphis. *Prof. Emer. of Patbol.* (4, 1937)

SCHMID, KARL, Ph.D. Massachusetts Gen. Hosp., Lovett Memorial Labs., Boston 14. *Assoc. Biochemist.* (2, 1954)

SCHMID, RUDI, M.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. in Med.* (4, 1958)

SCHMIDT, C. ROBERT, Ph.D., M.D. 407 Burns Bldg., Colorado Springs, Colo. *Surgeon.* (1, 1940)

SCHMIDT, CARL F., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Pharmacol.* (1, 1929; 3, 1924)

SCHMIDT, EMIL G., Ph.D. U. of Maryland Sch. of Med., Dept. of Biol. Chem., Baltimore 1. *Prof. and Head of Dept.* (2, 1951)

SCHMIDT, GERHARD, M.D. Boston Dispensary, 25 Bennet St., Boston, Mass. *Prof. of Biochem., Tufts U. Sch. of Med.* (2, 1939)

SCHMIDT, JOHN L., Ph.D., M.D. Abbott Labs. Res. Div., Dept. of Pharmacol., North Chicago, Ill. *Res. Pharmacologist.* (3, 1958)

SCHMIDT, L. H., Ph.D. Christ Hosp. Inst. of Med. Research, Cincinnati 19, Ohio. *Dir. of Research* (2, 1936; 3, 1946)

SCHMIDT-NIELSEN, BODIL, Dr.Odont., Dr.Phil. Duke U., Dept. of Zool., Durham, N. C. *Assoc. Res. Prof.; Estab. Invest. Amer. Heart Assn.* (1, 1949)

SCHMIDT-NIELSEN, KNUT, Dr.Phil. Duke U., Dept. of Zool., Durham, N. C. *Prof. of Physiol.* (1, 1949)

SCHMITT, FRANCIS OTTO, Ph.D. Massachusetts Inst. of Technology, Biol. Dept., Cambridge. *Inst. Prof.* (1, 1930)

SCHMITT, OTTO H., Ph.D. U. of Minnesota Sch. of Physics, Minneapolis 14. *Prof. of Biophysics (Zool. and Physics).* (1, 1947)

SCHNEIDER, BURCH H., Ph.D. State Coll. of Washington, Dept. of Animal Husbandry, Pullman. *Prof.* (5, 1952)

SCHNEIDER, CHARLES L., M.D., Ph.D. 22148 Michigan Ave., Dearborn, Mich. *Consultant, Obst. & Gynecol., Surg., Wayne Co. Gen. Hosp.* (1, 1949)

SCHNEIDER, HOWARD A., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (5, 1947)

SCHNEIDER, JOHN J., Ph.D., M.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Asst. Prof. Exptl. Med.* (2, 1950)

SCHNEIDER, JURG A., M.D. Chas. Pfizer & Co., Inc., Brooklyn 6, N. Y. *Dir., Macrobiol. Res.; Lect. in Pharmacol., Columbia U.* (1, 1956; 3, 1954)

SCHNEIDER, WALTER C., Ph.D. Natl. Cancer Inst., Lab. of Biol., NIH, Bethesda 14, Md. *Chemist.* (2, 1951)

SCHNEIERSON, S. STANLEY, M.D. Mt. Sinai Hosp., 1 East 100th St., New York City 29. *Attending Microbiologist.* (6, 1946)

SCHNEYER, LEON H., D.D.S., Ph.D. U. of Alabama Med. Ctr., Birmingham 3. *Assoc. Prof. of Physiol.; Assoc. Prof. of Dent.* (1, 1955)

SCHOENBORN, HENRY W., Ph.D. U. of Maryland, Dept. of Zool., College Park. *Prof.* (1, 1955)

SCHOEPFLE, GORDON M., Ph.D. Washington U. Sch. of Med., St. Louis, Mo. *Assoc. Prof. of Physiol.* (1, 1943)

SCHOLANDER, P. F., M.D., Ph.D. Scripps, Inst. of Oceanography, La Jolla, Calif. (1, 1947)

SCHOLLER, JEAN, Ph.D. Christ Hosp. Inst. of Med. Research, Cincinnati 19, Ohio. (3, 1956)

SCHREINER, GEORGE E., M.D. Georgetown U. Sch. of Med., Dept. of Med., Washington 7, D. C. *Asst. Prof. of Med.; Dir., Renal Clinic.* (1, 1958)

SCHREINER, OSWALD, Ph.D. 21 Primrose St., Chevy 15, Md. *Collaborator, USDA.* (2R, 1908)

SCHREK, ROBERT, M.D. VA Hosp., Hines, Ill. *Chief, Tumor Research Unit; Asst. Prof. of Pathol., Northwestern U.* (4, 1952)

SCHROEDER, HENRY A., M.D. West Brattleboro, Vt. (1, 1947)

SCHUBERT, JACK, Ph.D. Argonne Natl. Lab., Box 299, Lemont, Ill. *Sr. Chemist; Lect.-in-Chem., U. of Chicago.* (2, 1954)

SCHUBERT, MAXWELL, Ph.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Chem.* (3, 1947)

SCHUBERT, WALTER J., Ph.D. Fordham U. Coll. of Pharmacy, New York City 58. *Assoc. Prof. of Organic Chem. and Biochem.* (2, 1958)

SCHUCK, CECILIA, Ph.D. South Dakota State Coll., Div. of Home Econ., Brookings. *Prof. of Research.* (5, 1941)

SCHUELER, FRED W., Ph.D. Tulane U. Sch. of Med., Dept. of Pharmacol., New Orleans 12, La. *Prof. and Chmn.* (3, 1949)

SCHULMAN, MARTIN P., Ph.D. State U. of New York Sch. of Med., Dept. of Biochem., Syracuse 10. *Asst. Prof.* (2, 1954)

SCHULTZ, EDWIN WILLIAM, M.D. Stanford U., Stanford, Calif. *Prof. of Bacteriol. and Exptl. Pathol., Emer.* (4, 1927; 6R, 1928)

SCHULTZ, FRED H., JR., Ph.D. The Wander Co., 105 W. Adams St., Chicago, Ill. *Dir. of Research.* (3, 1948)

SCHULTZ, JULIUS, Ph.D. Hahnemann Med. Coll., Dept. of Biol. Chem., Philadelphia 2, Pa. *Assoc. Research Prof.* (2, 1955)

SCHULTZE, MAX O., Ph.D. U. of Minnesota, Dept. of Agric. Biochem., U. Farm, St. Paul 1. *Prof.* (2, 1938; 5, 1952)

SCHWAB, JOHN H., Ph.D. U. of North Carolina Sch. of Med., Dept. of Bacteriol., Chapel Hill. *Asst. Prof.* (6, 1956)

SCHWARTZ, IRVING L., M.D. Brookhaven Natl. Lab. Med. Dept., Upton, N. Y. *Sr. Scientist and Attending Physician.* (1, 1952)

SCHWARTZ, NEENA B., Ph.D. Michael Reese Hosp., Psychosomatic and Psychiatric Inst., Chicago 16, Ill. *Dir., Biol. Labs.* (1, 1956)

SCHWARTZ, SAMUEL, M.D. U. of Minnesota, Dept. of Med., Minneapolis 14, *Assoc. Prof. of Exptl. Med.* (2, 1952)

SCHWARTZ, WILLIAM B., M.D. New England Ctr. Hosp., Boston 11, Mass. *Assoc. Prof. of Med., Tufts U. Sch. of Med.* (1, 1957)

SCHWARZ, KLAUS, M.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Sec. on Exptl. Liver Diseases.* (2, 1958; 5, 1958)

SCHWEET, RICHARD S., Ph.D. City of Hope Med. Ctr., Dept. of Biochem., Duarte, Calif. *Chief, Sec. of Cardiac Chem.* (2, 1956)

SCHWEIGERT, B. S., Ph.D. U. of Chicago, Chicago 37, Ill. *Assoc. Prof. Biochem; Dir., Res., Amer.*

Meat Inst. Fndn. (2, 1949; 5, 1950)

SCHWEIZER, MALVINA, Ph.D. New York U., Washington Square Coll. of Arts and Sci., New York City. *Assoc. Prof. of Biol.* (1, 1944)

SCHWERDT, CARLTON E., Ph.D. Stanford U., Dept. of Med. Microbiol., Stanford, Calif. *Assoc. Prof.* (2, 1957)

SCHWERMA, HENRY, Ph.D., M.D. 6030 Sheridan Rd., Apt. 1304, Chicago 40, Ill. *Practicing Anesthesiologist.* (1, 1948)

SCHWERT, GEORGE W., Ph.D. Duke U. Sch. of Med., Dept. of Biochem., Durham, N. C. *Prof.* (2, 1949)

SCHWIMMER, SIGMUND, Ph.D. USDA, Western Regional Res. Lab., Albany 10, Calif. *Chemist, Vegetable Processing Div.* (2, 1947)

SCOTT, CHARLES COVERT, Ph.D., M.D. Warner-Lambert Research Inst., Morris Plains, N. J. *Vice Pres. for Basic Sciences.* (3, 1945)

SCOTT, DAVID ALYMER, Ph.D. Connaught Labs., U. of Toronto, Toronto 5, Canada. *Research Member.* (2R, 1935)

SCOTT, DONALD, JR., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof., Dept. of Physiol.; Assoc., Dept. of Neurol.* (1, 1950)

SCOTT, DWIGHT B., McNAIR, Ph.D. 421 Station Ave., Glenside, Pa. *Asst. Prof. of Physiol., U. of Pa.* (2, 1958)

SCOTT, EDWARD M., Ph.D. Arctic Health Research Ctr., Box 960, Anchorage, Alaska. *Biochemist.* (2, 1954)

SCOTT, ERNEST L., Ph.D. 64 South St., Bogota, N. J. *Assoc. Prof. Emer. of Physiol., Columbia U.* (1R, 1914; 2R, 1915)

SCOTT, H. M., Ph.D. U. of Illinois, Urbana. *Prof. of Animal Sci.* (5, 1956)

SCOTT, JAMES K., M.D. U. of Rochester, Atomic Energy Proj., Rochester 20, N. Y. *Chief Patbol. Sec., AEC; Assoc. in Patbol.* (4, 1949)

SCOTT, JOHN C., Ph.D. Hahnemann Med. Coll., Dept. of Physiol., Philadelphia 2, Pa. *Prof. and Head of Dept.* (1, 1936)

SCOTT, K. G., Ph.D. U. of California Sch. of Med., San Francisco 22. *Dir., Radioactivity Res. Ctr.; Prof. of Exptl. Radiol.* (3, 1956)

SCOTT, MILTON LEONARD, Ph.D. Cornell U., Rice Hall, Ithaca, N. Y. *Prof. of Animal Nutrition and Poultry Husbandry.* (2, 1952; 5, 1954)

SCOTT, V. BROWN, Ph.D., M.D. Inlow Clinic, Shelbyville, Ind. *Internist, Div. of Med.* (1, 1941)

SCOTT, W. W., Ph.D., M.D. Johns Hopkins Hosp., Brady Urological Inst., Baltimore 5, Md. *Urologist-in-Charge; Prof. of Urol.* (1, 1943)

SCOULAR, FLORENCE I., Ph.D. North Texas State Coll., Denton. *Dean, Sch. of Home Econ.; Prof. of Home Econ.* (5, 1958)

SCOW, ROBERT O., M.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Sr. Surgeon, USPHS.* (1, 1953)

SCRIMSHAW, NEVIN S., Ph.D., M.D. INCAP, Guatemala City, Guatemala. *Dir.; Reg. Advisor in Nutr., Pan Amer. Sanitary Bur., WHO.* (1, 1955; 5, 1949)

SCUDI, JOHN V., Ph.D. Wallace and Tiernan, Inc., Box 178, Newark 1, N. J. *Dir., Pharmaceutical Research.* (2, 1942; 3, 1950; 5, 1945)

SEAGER, LLOYD D., M.D. U. of Arkansas Sch. of Med., Dept. of Physiol. and Pharmacol., Little Rock. *Prof. and Head of Dept.* (3, 1939)

SEAMAN, GERALD R., Ph.D. U. of Texas Med. Br., Galveston. *Assoc. Prof. of Physiol.* (1, 1952)

SEASTONE, C. V., M.D. U. of Wisconsin Med. Sch., Dept. of Med. Microbiol., Madison 6. *Prof. and Chmn.* (6, 1939)

SEAY, PATRICK H., Ph.D. The Upjohn Co., Dept. of Pharmacol., Kalamazoo, Mich. *Research Pharmacologist.* (3, 1955)

SEBRELL, W. H., JR., M.D. Columbia U. Sch. of Public Health & Admin. Med., New York City 32. (2, 1938; 5, 1937)

SEEBERG, VICTOR P., Ph.D. Cutter Labs., Berkeley, Calif. *Assoc. Dir. of Research.* (3, 1953)

SEECOF, DAVID P., M.D. 1970 Daly Ave., New York City 60. (4, 1927)

SEED, JOHN C., M.D. The Wellcome Research Labs., Tuckahoe, N. Y. (3, 1952)

SEEGAL, BEATRICE CARRIER, M.D. Columbia U., Coll. of P. & S., New York City 32. *Assoc. Prof. of Microbiol.* (4, 1951; 6, 1949)

SEEGAL, DAVID, M.D. Goldwater Mem. Hosp., Columbia (First) Div., New York City 17. *Dir.; Prof. of Med., Columbia U.* (6, 1930)

SEEGERS, WALTER H., Ph.D. Wayne State U. Coll. of Med., Dept. of Physiol. and Pharmacol., Detroit 7, Mich. *Prof. and Chmn.* (1, 1947; 2, 1941)

SEEVERS, MAURICE HARRISON, Ph.D., M.D. U. of Michigan, Dept. of Pharmacol., Ann Arbor. *Prof. and Chmn. of Dept.* (1, 1933; 3, 1930)

SEGALOFF, ALBERT, M.D. Alton Ochsner Med. Fndn., 3503 Prytania St., New Orleans, La. *Dir. of Endocrine Research.* (4, 1946)

SEIBERT, FLORENCE B., Ph.D. U. of Pennsylvania, Henry Phipps Inst., 7th and Lombard Sts., Philadelphia. *Prof. of Biochem.* (2, 1925)

SEIBERT, RICHARD A., Ph.D. Baylor U., Coll. of Med., Dept. of Pharmacol., Houston 25, Tex. *Assoc. Prof.* (3, 1953)

SEIDELL, ATHERTON, Ph.D. 2301 Connecticut Ave., Washington 8, D. C. (2R, 1924)

SEIFTER, JOSEPH, M.D. Wyeth Inst. for Med. Research, P. O. Box 8299, Philadelphia 1, Pa. *Dir.* (3, 1940)

SEIFTER, SAM, Ph.D. Albert Einstein Coll. of Med., Dept. of Biochem., New York City 61. (2, 1946)

SELKURT, EWALD E., Ph.D. Indiana U. Sch. of Med., Indianapolis 7. *Prof. of Physiol.* (1, 1945)

SELL, HAROLD M., Ph.D. Michigan State U., Dept. of Agric. Chem., East Lansing. *Prof. (Research).* (2, 1954)

SELLE, WILBUR ARTHUR, Ph.D., M.D. U. of California Sch. of Med., Dept. of Biophysics, Los Angeles 24. *Prof.* (1, 1938)

SELLERS, ALVIN L., M.D. Cedars of Lebanon Hosp. Inst. for Med. Research, Los Angeles, Calif. *Research Assoc.* (1, 1956)

SELLERS, E. A., M.D., Ph.D. U. of Toronto, Dept. of Pharmacol., Toronto 5, Canada. (1, 1947)

SELYE, HANS, M.D., Ph.D. Inst. of Exptl. Med. and Surgery, U. of Montreal, Montreal, Canada. *Prof. and Dir.* (1, 1934)

SEMPLE, ROBERT E., Ph.D. Queen's U., Dept. of Physiol., Kingston, Canada. *Assoc. Prof.* (1, 1955)

SENDROY, JULIUS, JR., Ph.D. Naval Med. Research Inst., Natl. Naval Med. Ctr., Bethesda 14, Md. *Chief Chemist.* (1, 1954; 2, 1928)

SERAYDARIAN, MARIA W., Ph.D. VA Ctr., Los Angeles, Calif. *Estab. Invest., Los Angeles County Heart Assn.* (1, 1955)

SEVAG, M. G., Ph.D. U. of Pennsylvania Sch. of Med., Microbiol. Dept., Philadelphia 4. *Assoc. Prof.* (2, 1949; 6, 1941)

SEVERINGHAUS, ELMER L., M.D.. Columbia U., Inst. of Nutr. Sci., New York City 32. *Prof., Pub. Health Nutr.* (2, 1923; 5, 1939)

SEVERINGHAUS, JOHN W., M.D. U. of California Hosp., Cardiovascular Research Inst., San Francisco 22. (1, 1956)

SEVY, ROGER WARREN, Ph.D., M.D. Temple U. Sch. of Med., Dept. of Pharmacol., Philadelphia 40, Pa. *Prof. and Head.* (1, 1955)

SHACK, JOSEPH, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1954)

SHADLE, OSCAR W., M.D. U. of Louisville Sch. of Med., Louisville 2, Ky. *Assoc. Prof. of Physiol.* (1, 1955)

SHAFFER, C. BOYD, Ph.D. American Cyanamid Co., 30 Rockefeller Plaza, New York City 20. *Chief Indust. Toxicologist.* (3, 1950)

SHAFFER, MORRIS F., Ph.D. Tulane U. Sch. of Med., Dept. of Microbiol., New Orleans, La. *Prof. and Dept. Chmn.* (4, 1939; 6, 1937)

SHAFFER, PHILIP A., Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. Emer. of Biol. Chem.* (2, 1906)

SHANBERGE, JACOB N., M.D. Harvard Med. Sch., Boston 15, Mass. *Instr. in Pathol.* (4, 1958)

SHANES, ABRAHAM M., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Biophysicist.* (1, 1946; 3, 1958)

SHANK, ROBERT E., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Danforth Prof. of Prev. Med. and Head of Dept.* (2, 1947)

SHANNON, JAMES A., Ph.D., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Dir.* (1, 1933; 3, 1945)

SHAPIRO, HERBERT, Ph.D. Hosp. of U. of Pennsylvania, Philadelphia 4. *Asst. Prof. of Physical Med., Grad. Sch. of Med.* (1, 1937)

SHAPIRO, JOHN L., M.D. Vanderbilt U. Sch. of Med., Nashville, Tenn. *Prof. of Pathol.* (4, 1956)

SHARE, LEONARD, Ph.D. Western Reserve U. Sch. of Med., Dept. of Physiol., Cleveland 6, Ohio. *Asst. Prof.* (1, 1956)

SHARPLESS, GEORGE R., Sc.D. American Cyanamid Co., Research Div., Pearl River, N. Y. (5, 1942)

SHAUGHNESSY, HOWARD J., Ph.D. U. of Illinois, Dept. of Public Health, Chicago 12. *Prof. and Head; Dir. Labs., Ill. Health Dept.* (6, 1955)

SHAW, ELLIOTT, Ph.D. Tulane U., Dept. of Biochem., New Orleans, La. (2, 1954)

SHAW, J.C., Ph.D. U. of Maryland, Dept. of Dairy Husbandry, College Park. *Prof.* (1, 1947)

SHAW, JAMES H., Ph.D. Harvard Sch. of Dental Med., Boston 15, Mass. *Assoc. Prof. of Biol. Chem.* (5, 1948)

SHAW, KENNETH N. F., Ph.D. California Inst. of Technology, Gates & Crellin Labs. of Chem., Pasadena. *Sr. Research Fellow.* (2, 1957)

SHAW, MYRTLE, Ph.D. 11 S. Lake Ave., Albany, N. Y. *Assoc. Bacteriologist, Div. of Labs. and Research, N. Y. State Dept. of Health.* (6, 1937)

SHAY, HARRY, M.D. Temple U. Sch. of Med., Philadelphia, Pa. *Dir. of Fels Research Inst. and Prof. of Clin. Med.* (1, 1944)

SHEAR, MURRAY J., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Chem. Pharmacol.* (2, 1930; 3, 1953)

SHEARD, CHARLES, Ph.D. P. O. Box 543, Rochester, Minn. *Prof. & Dir. Emer. Biophys. & Physiol. Optics, Mayo Fndn. & Clin.* (1, 1925)

SHECHMEISTER, I. L., Ph.D. Southern Illinois U., Carbondale. *Assoc. Prof. of Microbiol.* (6, 1951)

SHEEHAN, DONAL, M.D., D.Sc. New York U. Bellevue Med. Ctr., New York City 16. (1, 1938)

SHEFFNER, A. LEONARD, Ph.D. Mead Johnson & Co., Dept. of Nutritional and Biochem. Research. Evansville 21, Ind. *Group Leader.* (2, 1954)

SHELESNYAK, M. C., Ph.D. Weizmann Inst. of Sci., Dept. of Exptl. Biol., Rehovoth, Israel. *Assoc.* (1, 1948)

SHELLEY, WALTER B., Ph.D., M.D. U. of Pennsylvania Hosp., Philadelphia 4. *Prof. of Dermatol.* (1, 1946)

SHELOKOV, ALEXIS I., M.D. Natl. Inst. of Allergy & Infectious Diseases, NIH, Bethesda 14, Md. *Acting Dir., Middle America Res. Unit.* (6, 1958)

SHELTON, EMMA, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biologist.* (4, 1957)

SHEMIN, DAVID, Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Biochem.* (2, 1944)

SHEPARD, CHARLES C., M.D. Public Health Service, P. O. Box 61, Montgomery 1, Ala. *Med. Dir.* (6, 1951)

SHEPARD, RICHARD H., M.D. Johns Hopkins U. Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Environ. Med.; Asst. Prof. Med.* (1, 1956)

SHEPPARD, C. W., Ph.D. U. of Tennessee, Div. of Physiol., Memphis 3. *Assoc. Prof.* (1, 1950)

SHEPPARD, FAY, M.S. U. of Oklahoma Sch. of Med., Oklahoma City. *Instr. in Biochem.* (2, 1936)

SHERMAN, F. G., Ph.D. Brown U., Dept. of Biol., Providence 12, R. I. *Prof.* (1, 1950)

SHERMAN, WILLIAM C., Ph.D. Chas. Pfizer & Co., Inc., Terre Haute, Ind. (5, 1949)

SHERROD, THEODORE R., Ph.D., M.D. U. of Illinois Coll. of Med., Chicago 12. *Assoc. Prof. of Pharmacol.* (3, 1952)

SHERRY, SOL, M.D. Washington U. Sch. of Med., St. Louis, 10, Mo. *Prof. of Med.* (1, 1955)

SHERWIN, CARL PAXSON, Sc.D., M.D., Dr.P.H. Whippleville, N. Y. *Consultant, Med., Hyde Mem. Hosp., Malone, N. Y.* (2R, 1917)

SHERWOOD, NOBLE P., Ph.D., M.D. 1801 Indiana St., Lawrence, Kans. *Consulting Pathologist, Workins Mem. Hosp.* (6, 1928)

SHERWOOD, THOMAS CECIL, Ph.D., M.D. Memorial Hosp., Orange, Tex. *Dir. of Anesthesia.* (1, 1938)

SHETTLES, LANDRUM B., Ph.D., M.D. Columbia U., Coll. of P. & S., New York City 32. *Asst. Prof. of Clin Obstetrics and Gynecol.* (1, 1946)

SHIDEMAN, FREDERICK E., Ph.D., M.D. U. of Wisconsin, Dept. of Pharmacol. and Toxicol., Madi-

son 6. *Prof. and Chmn.* (3, 1944)

SHILLING, CHARLES W., M.D. Atomic Energy Commission, Div. of Biol. and Med., Washington 25, D. C. *Dep. Dir.* (1, 1955)

SHILS, MAURICE E., M.D., Sc.D. 439 Sagamore Ave. Teaneck, N. J. *Intern, Second (Cornell) Div., Bellevue Hosp., New York City.* (5, 1950)

SHIMKIN, MICHAEL BORIS, M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief of Biometry Br.* (4, 1940)

SHINOWARA, GEORGE Y., Ph.D. Ohio State U., Coll. of Med., Div. of Chem. Pathol., Columbus 10. *Prof. and Chief of Div.* (2, 1949)

SHIPLEY, REGINALD A., M.D. VA Hosp., Cleveland 30, Ohio. *Dir., Radioisotope Unit.* (1, 1945)

SHIPLEY, ROBERT E., M.D. Lilly Lab. for Clin. Research, Indianapolis Gen. Hosp., Indianapolis, Ind. *Physiologist.* (1, 1945)

SHIRLEY, RAY L., Ph.D. U. of Florida, Nutrition Lab., Gainesville. *Prof. of Animal Husbandry and Nutrition.* (5, 1957)

SHIVE, WILLIAM, Ph.D. U. of Texas, Dept. of Chem., Austin. *Prof.* (2, 1948)

SHLAER, SIMON, Ph.D. Box 1663, Los Alamos, N. Mex. *Staff Member, Los Alamos Sci. Lab.* (1, 1938)

SHOCK, NATHAN W., Ph.D. Baltimore City Hosps., Baltimore 24, Md. *Chief, Gerontol. Br., Natl. Insts. of Health, Bethesda, Md.* (1, 1942)

SHOEMAKER, HAROLD A., Ph.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Prof. of Pharmacol.* (3, 1941)

SHOPE, RICHARD E., M.D. Ridge Road, Kingston, N. J. *Member, Rockefeller Inst.* (4, 1934)

SHORE, PARKHURST A., Ph.D. Natl. Heart Inst., Lab. of Chem. Pharmacol., NIH, Bethesda 14, Md. *Head, Sec. on Biochem. of Drug Action.* (3, 1956)

SHREEVE, WALTON W., M.D., Ph.D. Brookhaven Natl. Lab., Upton, N. Y. *Scientist, Med. Dept.* (2, 1955)

SHRIGLEY, E. W., Ph.D., M.D. Indiana U. Med. Ctr., Dept. of Microbiol., Indianapolis 7. *Prof. and Chmn. of Dept.* (6, 1946)

SHUBIK, PHILIPPE, D.Phil., B.M.B.Ch. Chicago Med. Sch., Chicago 8, Ill. *Prof. and Dir., Div. of Oncol.* (4, 1956)

SHULMAN, SIDNEY, Ph.D. U. of Buffalo Sch. of Med., Dept. Bacteriol. & Immunol., Buffalo 14, N. Y. *Asst. Prof., Immunochem.* (2, 1953)

SHUSTER, LOUIS, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1938)

SHWARTZMAN, GREGORY, M.D. 230 E. 50th St., New York City. *Prof. (retired) of Microbiol., Columbia U.* (4, 1929; 6, 1930)

SICHEL, F. J. M., Ph.D. U. of Vermont Coll. of Med. Dept. of Physiol and Biophysics, Burlington. *Prof. and Chmn. of Dept.* (1, 1939)

SICKLES, GRACE M., B.A. 2201 Twelfth St., Troy, N. Y. *Assoc. Research Scientist, Div. of Labs. and Res., N. Y. State Dept. of Health.* (6, 1932)

SICKLES, GRETCHEN R., A.B. New York State Dept. of Health, Div. of Labs. and Res., Albany, N. Y. *Assoc. Bacteriologist.* (6, 1937)

SIEBENMANN, CHARLES O., D.Eng. Connaught Med. Res. Labs., U. of Toronto, Toronto 5, Canada. *Res. Assoc.; Lect. in Microbiol.* (3, 1946)

SIEBENS, ARTHUR A., M.D. 205 N. Prospect Ave., Madison 5, Wis. (1, 1952)

SIEBERT, WALTER J., M.D. Lutheran Hosp., St. Louis, Mo. *Dir. of Labs.; Pathologist; Asst. Prof., Washington U.* (4, 1932)

SIEGMUND, OTTO HANNS, D.V.M. Merck Sharp & Dohme Research Labs., Animal Sci. Research, Rahway, N. J. (3, 1950)

SIEKEVITZ, PHILIP, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (2, 1954)

SIGEL, M. MICHAEL, Ph.D. U. of Miami, Sch. of Med., Coral Gables, Fla. *Prof., Bacteriol.; Dir., Virus Lab., Variety Children's Hosp.* (6, 1950)

SIGG, ERNEST BEAT, M.D. Geigy Chem. Corp., Dept. of Pharmacol., Ardsley, N. Y. *Chief Pharmacologist.* (1, 1957)

SIKOV, MELVIN R., Ph.D. Wayne U. Coll. of Med., Detroit 7, Mich. *Asst. Prof. of Radiobiol.* (4, 1958)

SILBER, ROBERT H., Ph.D. Merck Inst., Rahway, N. J. *Assoc. Dir. of Inst. and Dir. of Endocrinol.* (2, 1948)

SILBERBERG, MARTIN, M.D. 18 S. Kingshighway, St. Louis 8, Mo. *Assoc. Prof. of Patbol., Washington U.; City Hosp.* (4, 1944)

SILBERBERG, RUTH, M.D. 18 S. Kingshighway, St. Louis 8, Mo. *Assoc. Prof. of Patbol., Washington U.; City Hosp.* (4, 1944)

SILVERMAN, MILTON, Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Sr. Scientist.* (2, 1950)

SILVERMAN, MYRON S., Ph.D. U. S. Naval Radiol. Defense Lab., San Francisco 24, Calif. *Prin. Bacteriologist.* (6, 1957)

SILVETTE, HERBERT, Ph.D. "Low Gear," Stanardsville, Va. *Visiting Prof. of Theoretical Pharmacol., Med. Coll. of Virginia.* (1, 1933; 3, 1940)

SIMMONDS, SOFIA, Ph.D. Yale U. Sch. of Med., Dept. of Biochem., New Haven 11, Conn. *Assoc. Prof.* (2, 1948)

SIMMONS, DANIEL HAROLD, M.D., Ph.D. VA Ctr., Los Angeles 25, Calif. *Assoc. Prof. Med. and Physiol., UCLA Med. Ctr.* (1, 1957)

SIMON, ERIC J., Ph.D. New York Hosp., New York City 21. *Research Assoc. of Biochem., Cornell U. Med. Coll.* (2, 1957)

SIMONDS, JAMES P., Ph.D., M.D. 2150 Lincoln Park West, Chicago 14, Ill. *Prof. Emer., Patbol., Northwestern U.* (4, prior to 1920)

SIMONSEN, DAISY G., Ph.D. City of Hope Med. Ctr., Dept. of Biochem., Duarte, Calif. *Research Assoc.* (2, 1949)

SIMONSON, ERNST, M.D. U. of Minnesota, Dept. of Ophthalmol., Minneapolis 14. *Assoc. Prof. of Physiol. Ophthalmol.* (1, 1941)

SIMPSON, MELVIN V., Ph.D. Yale U. Sch. of Med., Dept. of Biochem., New Haven, Conn. *Assoc. Prof.* (2, 1955)

SIMPSON, MIRIAM E., Ph.D., M.D. U. of California, Berkeley. *Prof. of Anatomy.* (1, 1946)

SINCLAIR, HUGH, M.D. U. of Oxford, Oxford, England. *Dir., Lab. of Human Nutrition.* (5, 1953)

SINEX, F. MAROTT, Ph.D. Boston U. Sch. of Med., Dept. of Biochem., Boston, Mass. *Prof. and Chmn. of Dept.* (2, 1956)

SINGAL, SAM A., Ph.D. Med Coll. of Georgia, Augusta. *Prof. of Biochem.* (2, 1948; 5, 1955)

SINGER, IRA, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Research Assoc.* (4, 1956)

SINGER, LEON, Ph.D. U. of Minnesota, Dept. of Physiol. Chem., Millard Hall, Minneapolis. *Asst. Prof.* (2, 1957)

SINGER, S. JONATHAN, Ph.D. Yale U., New Haven, Conn. *Assoc. Prof. of Physical Chem.* (2, 1957)

SINGER, THOMAS P., Ph.D. Edsel B Ford Inst. for Med. Research, Henry Ford Hosp., Detroit 2, Mich. *Head, Enzyme Div.* (2, 1948)

SINGHER, H. O., Ph.D. Ortho Research Fndn., Raritan, N. J. *Dir., Div. of Biochem.* (1, 1956)

SINSHEIMER, ROBERT L., Ph.D. California Inst. of Technology, Div. of Biol., Pasadena 4. *Prof. of Biophysics.* (2, 1953)

SIROTA, JONAS H., M.D. 120 North 4th St., San Jose 12, Calif. (1, 1951)

SIZER, IRWIN W., Ph.D. Massachusetts Inst. of Technology, Cambridge. *Prof. of Biochem.; Head, Dept. of Biol.* (1, 1944; 2, 1955)

SJOERDSMA, ALBERT, M.D., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Sr. Invest.* (3, 1957)

SKELTON, F. R., M.D., Ph.D. Louisiana State U. Sch. of Med., New Orleans. *Assoc. Prof. Pathol.; Dir., Urban Maes Res. Fndn. and Lab.* (4, 1954)

SKINNER, CHARLES G., Ph.D. U. of Texas, Dept. of Chem., Austin. *Res. Scientist, Clayton Fndn. Biochem. Inst.* (2, 1958)

SKINNER, JOHN TAYLOR, Ph.D. Tennessee Polytech. Inst., Dept. of Chem., Cookeville. (2, 1946)

SKIPPER, HOWARD E., Ph.D. Southern Research Inst., 2000-9th Ave., So., Birmingham Ala. *Asst. Dir.* (2, 1953)

SKOOG, FOLKE, Ph.D. U. of Wisconsin, Dept. of Botany, Madison 6. *Prof.* (2, 1956)

SLATER, IRWIN H., M.D. Lilly Research Labs., Indianapolis 6, Ind. (3, 1951)

SLATOR, WILLIAM, JR., Ph.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Assoc. Prof. of Biophysics in Physiol.* (1, 1956)

SLEIN, MILTON W., Ph.D. 102 Norva Ave., Frederick, Md. *Biochemist, Fort Detrick.* (2, 1953)

SLOANE, NATHAN H., Ph.D. Mellon Inst., Pittsburgh, Pa. *Sr. Fellow.* (2, 1954)

SLOMKA, MYRON B., Ph.D. U. of Texas Southwestern Med. Sch., Dallas. *Asst. Prof. of Pharmacol.* (3, 1955)

SLOVITER, HENRY A., Ph.D., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Assoc. Prof.* (2, 1954)

SMADEL, JOSEPH EDWIN, M.D. Natl. Insts. of Health, Bethesda 14, Md. *Assoc. Dir.* (4, 1940; 6, 1937)

SMALL, JAMES C., M.D. 101 S. 39th St., Philadelphia 4, Pa. (4, 1927)

SMETANA, HANS F., M.D. Armed Forces Inst. of Pathol., Washington 25, D. C. (4, 1934)

SMITH, ALBERT G., M.D. VA Hosp., Durham, N. C. *Asst. Chief of Clin. Labs.* (4, 1958)

SMITH, ARTHUR H., Ph.D. Wayne State U. Coll. of Med., Dept. of Physiol. Chem., Detroit 7, Mich. *Prof. and Head of Dept.* (2, 1921; 5, 1933)

SMITH, AUSTIN EDWARD, M.D. American Med. Assn. 535 N. Dearborn St., Chicago, Ill. *Editor, J.A.M.A.* (3, 1942)

SMITH, C. A., Ph.D. 82 N. Summit St., Bergenfield, N. J. (1R, 1921)

SMITH, CARL C., Ph.D. Christ Hosp., Inst. of Med. Res., Cincinnati 19, Ohio. *Res. Assoc.; Asst. Prof., U. of Cincinnati.* (3, 1951)

SMITH, CEDRIC M., M.D. U. of Illinois Coll. of Med., Dept. of Pharmacol., Chicago. *Asst. Prof.* (3, 1955)

SMITH, DAVID T., M.D. Duke Hosp., Durham, N. C. *Prof. of Microbiol. and Assoc. Prof. of Med.* (5, 1943; 6, 1949)

SMITH, DIETRICH CONRAD, Ph.D. U. of Maryland Sch. of Med., Baltimore 1. *Prof. of Physiol.; Assoc. Dean.* (1, 1937)

SMITH, DONN L., Ph.D., M.D. U. of Colorado Sch. of Med., Dept. of Pharmacol., Denver 20. *Assoc. Prof.* (3, 1953)

SMITH, DOUGLAS E., Ph.D. Argonne Natl. Lab., P. O. Box 299, Lemont, Ill. *Assoc. Physiologist.* (1, 1947)

SMITH, DURWOOD JAMES, M.D. U. of Vermont Coll. of Med., Burlington. *Prof. of Pharmacol.* (1, 1952; 3, 1955)

SMITH, EDWIN L., Ph.D. U. of Texas Dental Br., Dept. of Physiol., Houston. *Prof. and Chmn. of Dept.* (1, 1948)

SMITH, ELINOR VAN DORN, Ph.D. 56 Middle St., Hadley, Mass. *Prof. of Bacteriol., Smith Coll.* (6, 1940)

SMITH, ELIZABETH R. B., Ph.D. 4323 Murdock Mill Rd., N. W., Washington 16, D. C. (2, 1938)

SMITH, EMIL L., Ph.D. U. of Utah Sch. of Med., Salt Lake City 1. *Prof. of Biochem.; Research Prof. of Med.* (2, 1946)

SMITH, ERMA A., Ph.D., M.D. VA Hosp., Cleveland 30, Ohio. *Physical Med.* (1, 1928)

SMITH, FALCONER, Ph.D. 9703 Cedar Lane, Bethesda, Md. *Sr. Scientist, PHS; Research in Radiobiol.* (1, 1950)

SMITH, FREDERICK G., Ph.D. Iowa State Coll., Botany and Chem. Depts., Ames. *Prof.* (2, 1954)

SMITH, GRANT NEWEY, Ph.D. Dow Chemical Co., Agric. Research Lab., Midland, Mich. *Biochemist.* (2, 1953)

SMITH, H. P., M.D. Columbia U., Coll of P. & S., New York City 32. *Delafield Prof. of Pathol.* (1, 1937; 4, 1925)

SMITH, HOMER W., Sc.D. New York U.-Bellevue Med. Ctr., New York City 16. *Prof. of Physiol.* (1, 1923; 2, 1930)

SMITH, JAMES JOHN, Ph.D., M.D. Marquette U. Sch. of Med., Milwaukee 3, Wis. *Prof. of Physiol.* (1, 1957)

SMITH, JANICE M., Ph.D. U. of Illinois, Dept. of Home Econ., Urbana. *Prof. of Nutrition and Head of Dept.* (5, 1947)

SMITH, JAY A., Ph.D. Chicago Med. Sch., Dept. of Physiol., Chicago 12, Ill. *Assoc. Prof.* (1, 1951)

SMITH, JOHN R., M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Assoc. Prof. of Med.* (1, 1947)

SMITH, LAWRENCE WELD, M.D. 1025 Fifth Ave., New York City 28. (4, 1927)

SMITH, LEE IRVIN, Ph.D. U. of Minnesota Sch. of

Chem., Div. of Organic Chem., Minneapolis 14. *Prof. and Chief.* (2, 1942)

SMITH, LUCILE, Ph.D. U. of Pennsylvania, Johnson Fndn., Philadelphia 4. *Asst. Prof. of Biophysics.* (2, 1956)

SMITH, MARGARET H.D., M.D. New York U. Sch. of Med., New York City 16. (6, 1953)

SMITH, PAUL K., Ph.D. George Washington U. Sch. of Med., Washington 5, D. C. *Prof. of Pharmacol.* (2, 1937; 3, 1937)

SMITH, PAUL W., Ph.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Prof. of Pharmacol.* (1, 1933; 3, 1951)

SMITH, PHILIP EDWARD, Ph.D. Stanford U., Dept. of Anatomy, Stanford, Calif. (1R, 1923)

SMITH, R. BLACKWE,, Jr., Ph.D. Med. Coll. of Virginia, Richmond 19. *Prof. of Pharmacol.; Pres.* (3, 1944)

SMITH, RALPH G., M.D., Ph.D. Food and Drug Admin., Washington 25, D. C. *Chief, New Drug Br.; Adj. Clin. Prof. Pharmacol., George Washington U.* (3, 1929)

SMITH, RICHARD T., M.D. U. of Texas, Southwestern Med. Sch., Dallas. *Assoc. Prof. of Pediatrics.* (4, 1957; 6, 1957)

SMITH, ROBERT E., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Physiol.* (1, 1953)

SMITH, SAM C., Ph.D. Research Corp., 405 Lexington Ave., New York City 17. *Secy. Williams-Waterman Fund.* (5, 1957)

SMITH, SEDGWICK E., Ph.D. Cornell U., Dept. of Animal Husbandry, Ithaca, N. Y. *Prof.* (5, 1945)

SMITH, SYBIL L., A.M. 1421-44th St., N. W., Washington 7, D. C. (5R, 1940)

SMITH, THOMAS C., Ph.D. Boston U. Sch. of Med., Dept. of Pharmacol. & Exptl. Therap., Boston 18, Mass. *Asst. Prof.* (3, 1958)

SMITH, WILBUR KENNETH, M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N. Y. *Assoc. Prof. of Anatomy.* (1, 1939)

SMITH, WILLIE W., Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Physiologist.* (1, 1941)

SMITHBURN, KENNETH C., M.D. Rockefeller Fndn., 49 West 49th St., New York City 20. *Staff Member.* (6, 1937)

SMOLENS, JOSEPH, B.S. Children's Hosp., Philadelphia, Pa. *Assoc. Dir., Serum Exch. Div.; Asst. Prof. of Microbiol. U. of Pa.* (6, 1943)

SMYTHE, C. V., Ph.D. P. O. Box 219, Bristol, Pa. *Head of Biochem. Lab., Rohm & Haas Co.* (2, 1934)

SMYTHIES, JOHN RAYMOND, M.D., D.P.M. Galesburg State Res. Hosp., Galesburg, Ill. *Psychiatrist II and Prin. Invest.* (3, 1958)

SNAPE, WILLIAM J., M.D. 573 Stevens St., Camden, N. J. (1, 1948)

SNEDECOR, JAMES G., Ph.D. U. of Massachusetts, Amherst. *Prof. of Physiol.* (1, 1957)

SNELL, ALBERT M., M.D. Palo Alto Clinic, 750 Northampton Ave., Palo Alto, Calif. (4, 1930)

SNELL, ESMOND E., Ph.D. U. of California, Dept. of Biochem., Berkeley 4. *Prof.* (2, 1942; 5, 1946)

SNIDER, RAY S., Ph.D. Northwestern U. Sch. of Med., Chicago 11, Ill. *Prof. of Anatomy.* (1, 1949)

SNOKE, JOHN E., Ph.D. U. of California Med. Ctr., Dept. of Physiol. Chem., Los Angeles 24. *Asst. Prof.* (2, 1954)

SNYDER, CHARLES D., Ph.D. 4709 Keswick Rd., Baltimore, Md. *Prof. Emer. of Exptl. Physiol. Johns Hopkins U.* (1R, 1907)

SNYDER, FRANKLIN FAUST, M.D. Boston Lying-In Hosp.. Boston 15, Mass. (1, 1936)

SNYDER, JOHN CRAYTON, M.D. Harvard Sch. of Public Health, Boston 15, Mass. *Prof. of Microbiol. and Dean of Faculty.* (6, 1950)

SNYDERMAN, SELMA E., M.D. New York U. Coll. of Med., Dept. of Pediatrics, New York City 16. *Assoc. Prof.* (5, 1953)

SOBEL, A. E., Ph.D. Jewish Hosp., Brooklyn 38, N. Y. *Head, Biochem.; Adj. Prof. Chem., Polytech. Inst.; Spec. Lect., State U. of N. Y.* (2, 1939)

SOBER, HERBERT A., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Head, Physiol. Chem. & Chromatography Sec.* (2, 1951)

SOBIN, SIDNEY S., M.D., Ph.D. 1200 N. State St., Los Angeles 33, Calif. *Research Prof. of Med., Coll. of Med. Evangelists* (1, 1949)

SOBOTKA, HARRY, Ph.D. Mt. Sinai Hosp., New York City 29. *Dir., Dept. of Chem.* (2, 1932; 1933)

SODERWALL, ARNOLD L., Ph.D. U. of Oregon, Dept. of Biol., Eugene. *Assoc. Prof.* (1, 1958)

SOKOLOFF, LEON, M.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Research Pathologist.* (4, 1956)

SOKOLOFF, LOUIS, M.D. Natl. Inst. of Mental Health, NIH, Bethesda 14, Md. *Chief, Sec. on Cerebral Metab.* (1, 1955)

SOLLMANN, TORALD, M.D. 14327 Superior Rd., Cleveland 18, Ohio. *Prof. Emer., Pharmacol., Western Reserve U.* (1R, 1902; 2R, 1906; 3, 1908)

SOLOMON, A. K., Ph.D. Harvard Med. Sch., Biophysical Lab., Boston 15, Mass. *Assoc. Prof. of Biophysics.* (1, 1952)

SOLOTOROVSKY, MORRIS, Ph.D. Rutgers, The State U., New Brunswick, N. J. *Prof. of Bacteriol.* (6, 1946)

SOMMERS, SHELDON C., M.D. Massachusetts Mem. Hosp., Boston. *Pathologist.* (4, 1954)

SOMOGYI, MICHAEL, Ph.D. 216 S. Kingshighway, St. Louis, Mo. *Biochemist, Jewish Hosp. of St. Louis.* (2R, 1927)

SONENBERG, MARTIN, M.D., Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc.* (2, 1956)

SONNENSCHEIN, RALPH R., M.D., Ph.D. U. of California Sch. of Med., Los Angeles 24. *Assoc. Prof. of Physiol.* (1, 1950)

SOODAK, MORRIS, Ph.D. Brandeis U. Sch. of Sci., Waltham 54, Mass. *Visiting Asst. Prof. of Biol. and Biochem.* (2, 1955)

SOROF, SAM, Ph.D. Inst. for Cancer Research, Fox Chase, Philadelphia 11, Pa. *Assoc. Member.* (2, 1955)

SOSKIN, SAMUEL, M.D., Ph.D. 9735 Wilshire Blvd., Beverly Hills, Calif. *Assoc. Clin. Prof. of Med., U. of California.* (1, 1930; 5, 1933)

SOURKES, THEODORE L., Ph.D. Allan Mem. Inst. of Psychiatry, 1025 Pine Ave., W., Montreal 2, Canada. *Asst. Prof. Biochem., McGill U.* (2, 1958)

SPAET, THEODORE H., M.D. Montefiore Hosp., New York City 67. *Dir. of Hematol.; Assoc. Prof.*

of Pathol., Columbia U. (1, 1956)

SPARGO, BENJAMIN H., M.D. U. of Chicago, Dept. of Pathol., Chicago 37, Ill. *Asst. Prof.* (4, 1956)

SPAULDING, EARLE H., Ph.D. Temple U., Sch. of Med., Dept. of Microbiol., Philadelphia 40, Pa. *Prof. and Head of Dept.* (6, 1950)

SPEALMAN, C. R., Ph.D. Dept. of Commerce, Civil Aeronautics Admin., Washington 25, D. C. *Chief, Safety Projects Br.* (1, 1940)

SPECHT, HEINZ, Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Lab. Phys. Biol.; Scientist Dir., PHS.* (1, 1941)

SPECTOR, HARRY, Ph.D. Quartermaster Food and Container Inst., 1819 W. Pershing Rd., Chicago 9, Ill. *Chief, Nutrition Br.* (5, 1950)

SPEIRS, MARY, Ph.D. U. of Georgia, Athens. *Dean, School of Home Econ.* (5, 1949)

SPEIRS, ROBERT S., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Asst. Prof. of Anatomy.* (1, 1956)

SPENCER, JOSEPH N., Ph.D. Smith, Kline & French Labs., Pharmacol. Sec., Philadelphia 1, Pa. (3, 1951)

SPENCER, MERRILL P., M.D. Bowman Gray Sch. of Med., Winston-Salem, N. C. *Asst. Prof. of Physiol. and Pharmacol.* (1, 1954)

SPERLING, FREDERICK, Ph.D. Sperling Labs., 6815 N. 24th St., Arlington 13, Va. *Dir.* (3, 1953)

SPERRY, ROGER W., Ph.D. California Inst. of Technology, Pasadena. *Hixon Prof. of Psychobiol.* (1, 1945)

SPERRY, WARREN M., New York State Psychiatric Inst., New York City 32. *Prof. of Biochem. Columbia U.* (2, 1929)

SPICER, SAMUEL S., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Med. Dir., PHS.* (1, 1950)

SPIEGEL, ERNEST A., M.D. Temple U. Sch. of Med., Dept. of Exptl. Neurol., Philadelphia, Pa. *Prof. and Head of Dept.* (1, 1936)

SPIEGEL-ADOLF, MONA, M.D. Temple U. Sch. of Med., Philadelphia, Pa. *Prof. of Colloid Chem.* (2, 1933)

SPIEGELMAN, SOL, Ph.D. U. of Illinois, Dept. of Bacteriol., Urbana. (1, 1946)

SPIES, TOM D., M.D. Hillman Hosp., Birmingham 3, Ala. *Dir. of Nutrition Clinic.* (3, 1941; 4, 1940; 5, 1938)

SPIKES, JOHN D., Ph.D. U. of Utah, Dept. of Exptl. Biol., Salt Lake City 12. *Prof. and Head.* (1, 1958)

SPINK, WESLEY W., M.D. U. of Minnesota Hosp., Minneapolis. *Prof. of Med., U. of Minn. Med. Sch.* (6, 1940)

SPIRTES, MORRIS A., M.D. Hahnemann Med. Coll., Philadelphia 2, Pa. *Assoc. Prof. of Pharmacol.* (3, 1955)

SPITZER, JOHN J., M.D. Hahnemann Med. Coll. and Hosp., Dept. of Physiol., Philadelphia 2, Pa. *Assoc. Prof.* (1, 1955)

SPIZIZEN, JOHN, Ph.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof.* (2, 1958; 6, 1957)

SPOHN, ADELAIDE, Ph.D. 7 New England Ave., Summit, N. J. *Nutritionist.* (5, 1933)

SPOOR, HERBERT J., Ph.D., M.D. 116 Parsons Blvd., Malba, N. Y. *Instr. in Med. (Dermatol.), Cornell U. Med. Coll.* (1, 1945)

SPRAGUE, JAMES MATHER, Ph.D. U. of Pennsylvania 4, Pa. *Prof.* (1, 1953)

SPRENT, JOHN F. A., D.Sc., Ph.D. U. of Queensland School of Veterinary Science, Brisbane, Australia. *Prof.* (6, 1955)

SPRINGER, GEORG F., M.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof. of Immunology in Med. and in Microbiol.* (6, 1956)

SPRINSON, DAVID B., Ph.D. Columbia U. Coll. of P. & S., Dept. of Biochem., New York City 32. *Prof.* (2, 1949)

SPROUL, EDITH E., M.D. Columbia U., Coll. of P. & S., Dept. of Pathol., New York City 32. *Assoc. Prof.* (4, 1941)

SPRUNT, DOUGLAS H., M.D. U. of Tennessee, Div. of Pathol. and Microbiol., Memphis. *Chmn.; Prof. of Pathol.* (4, 1934; 6, 1936)

SPURR, GERALD B., Ph.D. U. of Tennessee Med. Units, Inst. of Clin. Invest., Memphis 3. *Asst. Prof. of Clin. Physiol.* (1, 1957)

SQUIBB, ROBERT L., Ph.D. Rutgers, The State U., Dept. of Poultry Sci., New Brunswick, N. J. *Chmn.* (5, 1953)

SQUIRES, RUSSELL DILL, M.D. U. of Pennsylvania Sch. of Med., Dept. of Physiol., Philadelphia 4. *Asst. Prof.* (1, 1957)

STACY, RALPH W., Ph.D. Ohio State U., Div. of Biophysics, Dept. of Physiol., Hamilton Hall, Columbus 10. *Assoc. Prof.* (1, 1949)

STADIE, WILLIAM C., M.D. U. of Pennsylvania, 817 Maloney Clinic, Philadelphia 4, Pa. *Emer. Prof. of Res. Med.* (2R, 1922)

STADTMAN, EARL R., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1951)

STADTMAN, THRESSA C., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1957)

STAHMANN, MARK A., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Prof.* (2, 1951)

STAMLER, FREDERIC W., M.D. State U. of Iowa, Iowa City. *Assoc. Prof. of Patbol.* (4, 1956)

STAMLER, JEREMIAH, M.D. Michael Reese Hosp., Med. Res. Inst., Cardiovas. Dept., Chicago, 16, Ill. *Asst. Dir.; Estab. Invest. Am. Heart Assn.* (1, 1951)

STANIER, ROGER Y., Ph.D. U. of California, Dept. of Bacteriol., Berkeley 4. *Prof.* (2, 1956)

STANLEY, WENDELL M., Ph.D., ScD. U. of California, Virus Lab., Berkeley 4. *Dir.; Prof. of Virol.* (2, 1936; 6, 1957)

STANNARD, JAMES NEWELL, Ph.D. U. of Rochester Sch. of Med., Rochester 20, N.Y. *Assoc. Prof., Pharmacol. & Radiation Biol.* (1, 1938; 3, 1953)

STANSLY, PHILIP G., Ph.D. Detroit Inst. for Cancer Res., Detroit 1, Mich. *Res. Assoc.; Assoc. Prof. of Microbiol., Wayne State U.* (2, 1950)

STANTON, MEARL F., M.D. 11705 Viers Mill Rd., Silver Spring, Md. *Lab. of Patbol., Natl. Cancer Inst.* (4, 1957)

STAPLE, EZRA, Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof. of Biochem.* (2, 1957)

STARE, FREDRICK J., Ph.D., M.D. Harvard U., Dept. of Nutrition, Boston 15, Mass. *Prof. and Head of Dept.* (2, 1937; 5, 1942)

STARK, LAWRENCE, M.D. Yale U. Sch. of Med., Dept. of Int. Med., New Haven, Conn. *Asst. Prof.,*

Sec. of Neurol. (1, 1958)

STARR, ISAAC, M.D. Hosp. of U. of Pennsylvania, Philadelphia 4. *Hartzell Res. Prof. of Therap.* (1, 1929; 3, 1942)

STARY, ZDENKO, M.D., Ph.D. Warren State Hosp., Warren, Pa. (2, 1958)

STAUB, ALFRED, Ph.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Biochem.* (2, 1956)

STAVELY, HOMER E., Ph.D., D.Sc. Mead Johnson & Co., Evansville 21, Ind. *Dir., Natural Products Research.* (2, 1952)

STAVITSKY, ABRAM B., Ph.D., V.M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof. of Microbiol.* (6, 1950)

STAVRAKY, GEORGE W., M.D. U. of Western Ontario Med. Sch., London, Canada. *Prof. of Physiol.* (1, 1937; 3, 1944)

STEAD, EUGENE A., JR., M.D. Duke U., Dept. of Med., Durham, N.C. (1, 1945)

STEARNS, GENEVIEVE, Ph.D. State U. of Iowa Coll. of Med., Iowa City. *Research Prof. Emer. of Orthopedics.* (2, 1932; 5, 1937)

STEBBINS, ROBERT B., Ph.D. Merck Sharp & Dohme Research Labs., Merck & Co., Inc., West Point, Pa. *Res. Assoc. in Pathol.* (4, 1951)

STEELE, BETTY F., Ph.D. 1208 Henry St., Ann Arbor, Mich. (5, 1954)

STEELE, J. MURRAY, M.D. Goldwater Mem. Hosp., Welfare Is., New York City 17. *Prof. of Med., New York U.; Dir. of 3rd N.Y.U. Res. Serv.* (1, 1936)

STEELE, ROBERT, Ph.D. Brookhaven Natl. Lab., Biol. Dept., Upton, N.Y. *Biochemist.* (2, 1952)

STEELMAN, SANFORD L., Ph.D. Baylor U. Coll. of Med., Texas Med. Ctr., Dept. of Biochem., Houston. *Assoc. Prof.* (2, 1958)

STEENBOCK, HARRY, Ph.D., Sc.D. U. of Wisconsin, Biochem. Bldg., Madison 6. *Prof. of Biochem.* (2R, 1912; 5, 1933)

STEFANINI, M., M.D., D.T.M.&H. St. Elizabeth Hosp., Boston, Mass. *Dir., Stanton Labs.; Assoc. Prof., Med., Tufts U.* (1, 1952; 4, 1956; 6, 1956)

STEFFEE, C. HAROLD, M.D., Ph.D. Oak Ridge Inst. of Nuclear Studies, Med. Div., Oak Ridge, Tenn. *Chief of Pathol.* (4, 1950)

STEGGERDA, F. R., Ph.D. U. of Illinois, 416 Natural History Bldg., Urbana *Prof. of Physiol.* (1, 1934)

STEIGMAN, ALEX J., M.D. U. of Louisville Sch. of Med., Dept. of Pediatrics, Louisville 2, Ky. *Chmn. of Dept.* (6, 1949)

STEIGMANN, FREDERICK, M.D. 2044 Farwell Ave., Chicago 45, Ill. *Clin. Assoc. Prof. Med., U. of Ill.; Attend. Phys., Cook Co. Hosp.* (3, 1942)

STEIMAN, S. E., Ph.D., M.D. 23 Broad St., Lynn, Mass. (1, 1939)

STEIN, GEORGE J., Ph.D. USOM, American Embassy, Saigon, Vietnam. (6, 1947)

STEIN, S. N., M.D. Naval Med. Research Inst., Bethesda 14, Md. (1, 1953)

STEIN, WILLIAM H., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1946)

STEINBACH, H. BURR, Ph.D. U. of Chicago, Dept. of Zool., Chicago, Ill. *Prof.* (1, 1934)

STEINBERG, BERNHARD, M.D. Toledo Hosp. Inst. of Med. Res., Toledo, Ohio. *Dir.; Chief Pathologist & Dir. of Labs., Toledo Hosp.* (4, 1928; 6, 1946)

STEINBERG, DANIEL, M.D., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chief, Sec. on Metab.* (2, 1956)

STEINER, PAUL E., M.D., Ph.D. U. of Chicago, Chicago, Ill. *Prof. of Pathol.* (4, 1939)

STEINHARDT, JACINTO, Ph.D. 4004 Laird Pl., Chevy Chase 15, Md. *Dir., Operations Evaluation Group, Mass. Inst. of Technology.* (2, 1939)

STEINHAUS, ARTHUR H., Ph.D. 5315 Drexel Ave., Chicago, Ill. *Prof. of Physiol. and Dean, George Williams Coll.* (1, 1928)

STEINHAUS, JOHN E., M.D., Ph.D. Emory U. Sch. of Med., Anesthesia Sec., Atlanta 22, Ga. *Assoc. Prof.* (3, 1951)

STEINMAN, H. G., Ph.D. Natl. Inst. of Allergy and Infectious Diseases, Lab. of Clin. Invest., NIH, Bethesda 14, Md. *Chief, Sec. Biochem.* (2, 1954)

STEKOL, JAKOB A., Sc.D. Inst. for Cancer Research, Philadelphia 11, Pa. *Head, Dept. of Physiol. Chem. and Nutrition.* (2, 1936; 5, 1952)

STEPHENSON, EDWARD L., Ph.D. U. of Arkansas Coll. of Agric., Fayetteville. *Prof.* (5, 1958)

STEPTO, ROBERT C., M.D., Ph.D. Loyola U., Stritch Sch. of Med., Chicago, Ill. *Asst. Prof., Obstetrics and Gynecol.* (4, 1952)

STERN, JOSEPH R., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Assoc. Prof. of Pharmacol.* (2, 1952)

STERN, KURT, M.D. Mt. Sinai Med. Res. Fndn., Chicago, Ill. *Dir., Blood Ctr.; Assoc. Prof. Pathol., Chicago Med. Sch.* (4, 1957; 6, 1954)

STERNBERGER, LUDWIG A., M.D. Chem. Warfare Labs., Army Chemical Center, Md. *Acting Chief, Pathol. Br.* (6, 1954)

STETSON, CHANDLER A., M.D. New York U. Bellevue Med. Ctr., Dept. of Pathol., New York City 16. *Prof. and Chmn.* (4, 1958; 6, 1957)

STETTEN, DEWITT, JR., M.D., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. (2, 1944)

STETTEN, MARJORIE R., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Chemist (Biochem.)* (2, 1947)

STEVENS, CARL M., Ph.D. State Coll. of Washington, Dept. of Chem., Pullman. *Prof. of Biochem.* (2, 1951)

STEVENS, KINGSLEY M., M.D. Merck Sharp & Dohme Research Labs., West Point, Pa. *Research Assoc.* (6, 1955)

STEVENS, S. SMITH, Ph.D. Harvard U., Memorial Hall, Cambridge, Mass. *Prof. of Psychology; Dir. of Psychol. Labs.* (1, 1937)

STEVENSON, JAMES A. F., M.D.C.M. U. of Western Ontario, Dept. of Physiol., London, Canada. *Prof. and Head of Dept.* (1, 1950)

STEWART, DOROTHY R., Ph.D. Rockford Coll., Biol. Dept., Rockford, Ill. *Chmn.* (1, 1947)

STEWART, FRED W., M.D. Memorial Hosp., New York City 21. *Pathologist; Prof. of Pathol., Sloan-Kettering Div., Cornell U.* (4, 1928)

STEWART, HAROLD L., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Pathol. and Dept. of Pathol. Anatomy.* (4, 1936)

STEWART, PETER A., Ph.D. Emory U., Div. of Bas-

ic Health Sci., Physiol. Dept., Atlanta 22, Ga. *Asst. Prof.* (1, 1957)

STEWART, WELLINGTON B., M.D. Columbia U. Coll. of P. & S., New York City 32. *Assoc. Prof. of Pathol.* (4, 1950)

STICKNEY, J. CLIFFORD, Ph.D. West Virginia U. Med. Ctr., Morgantown. *Prof. of Physiol.* (1, 1944)

STIEBELING, HAZEL K., Ph.D. USDA, Agric. Research Serv., Washington 25, D.C. *Dir., Inst. of Home Econ.* (5, 1933)

STIER, THEODORE J. B., Ph.D. Indiana U. Sch. of Med., Bloomington. *Prof. of Physiol.* (1, 1938)

STILL, EUGENE U., Ph.D. Box 991, Sarasota, Fla. (1, 1929)

STILL, JOSEPH W., M.D. George Washington U. Sch. of Med., Washington 5, D.C. *Asst. Prof. of Physiol.* (1, 1956)

STIMMEL, BENJAMIN F., Ph.D. Rees-Stealy Clinic Research Fndn., 2001 Fourth Ave., San Diego, Calif. *Dir.* (2, 1947)

STOCK, AARON H., M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Assoc. Prof. of Microbiol.* (6, 1947)

STOCK, C. CHESTER, Ph.D. Sloan-Kettering Inst. for Cancer Res., New York City 21. *Assoc. Dir. and Chief, Div. of Exptl. Chemotherapy.* (2, 1952)

STOERK, HERBERT C., M.D. Merck Inst. for Therap. Research, Rahway, N. J. *Dir. of Exptl. Pathol.* (4, 1948)

STOESSER, ALBERT V., M.D., Ph.D. U. of Minnesota Med. Sch., Mayo Mem. Bldg., Minneapolis. *Prof. of Pediatrics.* (6, 1949)

STOHLMAN, EDWARD F., LL.B. Natl. Insts. of Health, Bethesda 14, Md. *Assoc. Pharmacologist.* (3, 1948)

STOHLMAN, FREDERICK, JR., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Research Hematologist.* (4, 1956)

STOKINGER, HERBERT E., Ph.D. PHS Occupational Health Field HQ, Cincinnati 2, Ohio. *Chief, Toxicologic Services.* (3, 1950; 6, 1947)

STOKSTAD, E. L. ROBERT, Ph.D. American Cyanamid Co., Research Div., Pearl River, N.Y. *Asst. Dir., Nutrition and Physiol.* (2, 1947; 5, 1942)

STOLAND, O. O., Ph.D. 1845 Leamard Ave., Lawrence, Kans. *Prof. of Physiol. Emer., U. of Kansas.* (1R, 1913)

STOLL, ALICE M., M.S. Naval Air Develop. Ctr., Johnsville, Pa. *Physiologist.* (1, 1955)

STOLL, ARTHUR, Ph.D. Sandoz, Inc., Basle 13, Switzerland. *Pres., Internatl. Union of Pure and Applied Chem.* (3, 1951)

STOLLERMAN, GENE HOWARD, M.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Assoc. Prof. of Med.* (6, 1957)

STONE, CLEMENT A., Ph.D. Merck Inst. for Therap. Res., West Point, Pa. *Dir. of Pharmacodynamics.* (3, 1954)

STONE, DAVID, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Res. Assoc.* (2, 1955)

STONE, GILBERT C. H. Ph.D. City College of New York, Dept. of Chem., New York City 31. *Assoc. Prof.* (2, 1950)

STONE, SANFORD H., Sc.D. Natl. Inst. of Allergy and Infectious Diseases, NIH, Bethesda 14, Md. *Lab. of Immunol.* (6, 1957)

STONE, WILLIAM E., Ph.D. U. of Wisconsin, Dept. of Physiol., Madison. *Assoc. Prof.* (1, 1945)

STONER, RICHARD D., Ph.D. Brookhaven Natl. Lab., Med. Dept., Upton, N.Y. *Scientist, Div. of Microbiol.* (4, 1957)

STORER, JOHN B., M.D. Jackson Mem. Lab., Bar Harbor, Maine. *Radiobiologist.* (4, 1952)

STORMONT, CLYDE JUNIOR, Ph.D. U. of California Sch. of Veterinary Med., Davis. *Assoc. Prof. of Vet. Sci.; Assoc. Serologist in Expt. Sta.* (6, 1958)

STORMONT, ROBERT T., Ph.D., M.D. American Med. Assn., 535 N. Dearborn St., Chicago, Ill. *Dir., Div. Therapy and Res.* (3, 1941)

STORVICK, CLARA A., Ph.D. Oregon State Coll. Sch. of Home Econ., Corvallis. *Prof. of Foods and Nutrition; Head of Home Econ. Res.* (5, 1947)

STOTZ, ELMER H., Ph.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. of Biochem.* (2, 1939)

STOWELL, ROBERT E., M.D., Ph.D. U. of Kansas Med. Ctr., Dept. of Pathol. and Oncol., Kansas City 12. *Prof. and Chmn.* (4, 1952)

STRASSMAN, MURRAY, Ph.D. Inst. for Cancer Res., Philadelphia 11, Pa. *Res. Assoc.* (2, 1958)

STRECKER, HAROLD J., Ph.D. Albert Einstein Coll. of Med., Dept. of Biochem., New York City 61. *Assoc. Prof.* (2, 1953)

STREHLER, BERNARD L., Ph.D. Natl. Insts. of Health, Gerontol. Br., Baltimore City Hosps., Baltimore 24, Md. (2, 1955)

STRENGTH, D. RALPH, Ph.D. St. Louis U., Dept. of Biochem., St. Louis, Mo. *Asst. Prof.* (2, 1956)

STRICKLAND, KENNETH P., Ph.D. U. of Western Ontario, Faculty of Med., Dept. of Biochem., London, Canada. *Asst. Prof.* (2, 1957)

STRITTMATTER, CORNELIUS F., Ph.D. Harvard Med. Sch., 25 Shattuck St., Boston 15, Mass. *Asst. Prof. of Biol. Chem.* (2, 1956)

STROMINGER, JACK L., M.D. Washington U. Sch. of Med., Dept. of Pharmacol., St. Louis 10, Mo. *Assoc. Prof.* (2, 1957; 3, 1956)

STRONG, FRANK M., Ph.D. U. of Wisconsin, Madison 6. *Prof. of Biochem.* (2, 1941)

STRONG, JACK P., M.D. Louisiana State U. Sch. of Med., New Orleans 12. *Asst. Prof. of Pathol.* (4, 1958)

STRUCK, HAROLD CARL, Ph.D. 528 N. Garfield Ave., Montebello, Calif. (1, 1940; 3, 1952)

STRUGHOLD, HUBERTUS, M.D., Ph.D. USAF, Sch. of Aviation Med., Randolph AFB, Tex. *Prof. of Space Med. and Advisor for Res.* (1, 1951)

STUART, CHARLES A., Ph.D. 372 Lloyd Ave., Providence, R.I. *Prof. of Biol., Brown U.* (6, 1935)

STUMPF, PAUL K., Ph.D. U. of California, Dept. of Agric. Biochem., Davis. *Prof. of Plant Biochem.* (2, 1950)

STURGIS, CYRUS CRESSEY, M.D. Simpson Mem. Inst., Ann Arbor, Mich. *Prof. of Med., U. of Michigan Hosp.* (4, 1927)

STURKIE, PAUL D., Ph.D. Rutgers, The State U., Coll. of Agric., Poultry Sci. Dept., New Brunswick, N. J. *Prof. of Poultry Physiol.* (1, 1948)

STURTEVANT, F. M., Ph.D. Smith, Kline & French Labs., Philadelphia 1, Pa. *Sr. Pharmacologist.*

(3, 1955)

STUTZMAN, J. W., Ph.D., M.D. Riker Labs., Inc., Northridge, Calif. *Vice Pres., Research.* (1, 1946; 3, 1948)

SUCKLING, E. E., M.Sc., D.E.E. State U. of New York Coll. of Med., Dept. of Physiol., Brooklyn 3. *Assoc. Prof.* (1, 1955)

SUGG, JOHN Y., Ph.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Bacteriol. and Immunol.* (6, 1938)

SULKIN, S. EDWARD, Ph.D. U. of Texas Southwestern Med. Sch., Dept. of Microbiol., Dallas. *Prof. and Chmn. of Dept.* (6, 1944)

SULLIVAN, MICHAEL XAVIER, Ph.D. Georgetown U. Grad. Sch., Washington 7, D.C. *Research Prof. of Chem. Emer.* (2R, 1909)

SUMMERSON, WILLIAM H., Ph.D. Chem. Warfare Labs., Army Chemical Center, Md. *Dir. of Research.* (2, 1942)

SUNAHARA, FRED AKIRA, Ph.D. Defence Research Med. Labs., Toronto, Canada. *Defence Sci. Serv. Officer.* (1, 1957)

SUNDE, M. L., Ph.D. U. of Wisconsin Coll. of Agric., Dept. of Poultry Husbandry, Madison 6. *Prof.* (5, 1955)

SUNDERMAN, F. WILLIAM, M.D., Ph.D. Jefferson Med. Coll., Philadelphia 7, Pa. *Dir., Div. of Metabolic Res. and Clin. Prof. of Med.* (2, 1931)

SUNDSTROEM, EDWARD S., M.D. 2491 Ellsworth St., Berkeley 4, Calif. *Prof. Emer., U. of California.* (2R, 1919)

SUNG, CHEN-YU, Ph.D. Chinese Acad. of Med. Sci., Dept. of Pharmacol., Peking, China. *Asst. Prof.* (3, 1953)

SURE, BARNETT, Ph.D. U. of Arkansas, Fayetteville. *Head of Dept. and Prof. of Agric. Chem.* (2R, 1923; 5R, 1933)

SURGENOR, DOUGLAS MacN., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Asst. Prof. of Biol. Chem.* (2, 1957)

SURTSHIN, ADOLPH, M.D. 19901 Nordhoff St., Northridge, Calif. (1, 1951)

SUSKIND, SIGMUND R., Ph.D. Johns Hopkins U., McCollum-Pratt Inst., Baltimore 18, Md. *Asst. Prof. of Biol.* (6, 1958)

SUTER, EMANUEL, M.D. U. of Florida Coll. of Med., Gainesville. *Prof. of Microbiol.* (6, 1954)

SUTHERLAND, EARL W., JR., M.D. Western Reserve U. Sch. of Med., Cleveland 6, Ohio. *Prof. and Dir. of Pharmacol.* (2, 1950; 3, 1957)

SUTHERLAND, GEORGE F., M.D. Maryland Dept. of Mental Hygiene, Baltimore 18, Md. *Chief, Div. of Psychiatric Educ. & Training.* (1, 1939)

SUTHERLAND, VIOLETTE CUTTER, Ph.D. U. of California Sch. of Med., Dept. of Pharmacol. & Exptl. Therap., San Francisco 22. *Asst. Prof.* (3, 1958)

SUTTON, T. SCOTT, Ph.D. Ohio State U., Coll. of Agric., Columbus. *Assoc. Dean.* (5, 1936)

SUTTON, WILLIAM B., Ph.D. Lilly Research Labs., Eli Lilly & Co., Indianapolis 6, Ind. *Enzymologist.* (2, 1957)

SVIRBELY, JOSEPH L., Ph.D. Robert A. Taft Sanitary Engin. Ctr., Cincinnati 26, Ohio. *Toxicologist.* (3, 1945)

SWAIN, HENRY H., M.D. U. of Michigan Med. Sch., Dept. of Pharmacol., Ann Arbor. *Asst. Prof.* (3, 1957)

SWAIN, ROBERT E., Ph.D. 634 Mirada Ave., Stanford, Calif. *Prof. Emer. of Chem., Stanford U.* (2R, 1909)

SWAN, H. J., M.B., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant in Physiol.* (1, 1954)

SWAN, ROY CRAIG, JR., M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Physiol.* (1, 1954)

SWANN, HOWARD G., Ph.D. U. of Texas Sch. of Med., Dept. of Physiol., Galveston. *Prof.* (1, 1940)

SWANSON, EDWARD E., D.Sc. 7920 Barlum Dr., Indianapolis 20, Ind. *Teacher of Bio-Assay, Butler U.* (3, 1956)

SWANSON, MARJORIE A., Ph.D. Bowman Gray Sch. of Med., Dept. of Biochem., Winston-Salem 7, N.C. *Assoc. Prof.* (2, 1950)

SWANSON, PEARL, Ph.D. Iowa State Coll., Ames. *Prof. of Nutrition; Asst. Dir., Agric. and Home Econ. Expt. Sta.* (5, 1933)

SWANSON, WILLIAM W., M.D. 2376 E. 71st St., Chicago, Ill. *Asst. Prof. of Pediatrics, Northwestern U.* (2, 1938)

SWEAT, MAX L., Ph.D. U. of Utah Coll. of Med., Dept. of Obstetrics and Gynecol., Salt Lake City. *Assoc. Research Prof.* (2, 1954)

SWEENEY, H. MORROW, Ph.D. 5608 Bull Creek Rd., Austin, Tex. (1, 1939)

SWELL, LEON, Ph.D. VA Ctr., Gen. Med. Research, Martinsburg, W. Va. *Chief Biochemist.* (2, 1957)

SWENDSEID, MARIAN E., Ph.D. U. of California, Los Angeles 24. *Assoc. Prof. of Nutrition and Physiol. Chem.* (2, 1950)

SWICK, ROBERT W., Ph.D. Argonne Natl. Lab., Div. of Med. and Biol. Research, P.O. Box 299, Lemont, Ill. *Assoc. Biochemist.* (2, 1957)

SWIFT, MARGUERITE N., M.S. U.S. Naval Radiol. Defense Lab., San Francisco 24, Calif., *Radiol. Biologist.* (1, 1956)

SWIFT, RAYMOND W., Ph.D. Pennsylvania State U., Dept. of Animal Nutrition, University Park. *Prof. and Head of Dept.* (5, 1934)

SWIM, H. EARLE, Ph.D. Western Reserve U., Cleveland 6, Ohio. *Asst. Prof. of Microbiol.* (2, 1958)

SWINGLE, WILBUR WILLIS, Ph.D. Princeton U., Princeton, N.J. *Henry Fairfield Osborn Prof. of Biol.* (1, 1924)

SWINYARD, EWART A., Ph.D. U. of Utah Salt Lake City. *Prof. Pharmacol., Coll. Pharmacy; Lect., Pharmacol., Coll. Med.* (3, 1948)

SYDENSTRICKER, V. P., M.D. VA Hosp., Forest Hills Div., Augusta, Ga. *Asst. Chief Med. Dir. of Research.* (5, 1944)

SYKES, JOSEPH F., Ph.D. USDA Dairy Husbandry Research Br., Beltsville, Md. *Physiologist.* (1, 1942)

SYVERTON, JEROME T., M.D. U. of Minnesota, Dept. of Bacteriol. and Immunol., Minneapolis. *Prof. and Head.* (4, 1940; 6, 1947)

SZEGO, CLARA M., Ph.D. U. of California, Los Angeles 24. *Assoc. Prof. of Zool.* (1, 1946)

SZENT-GYORGYI, ALBERT, Ph.D., M.D. Inst. for Muscle Research, Marine Biol. Lab., Woods Hole, Mass. *Research Dir.* (2, 1949)

SZENT-GYORGYI, ANDREW G., M.D. Inst. for Mus-

cle Research, Marine Biol. Lab., Woods Hole, Mass. *Estab. Invest., Amer. Heart Assn.* (1, 1956; 2, 1958)

SZEPSENWOL, JOSEL, M.D. U. of Puerto Rico Sch. of Med., San Juan 22. *Assoc. Prof. of Anatomy.* (1, 1948)

TABACHNICK, IRVING I. A., Ph.D. Schering Corp., Bloomfield, N. J. *Pharmacologist.* (3, 1958)

TABOR, CELIA WHITE, M.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. (3, 1958)

TABOR, HERBERT, M.D. Natl. Inst. of Arthritis & Metabolic Diseases, NIH, Bethesda 14, Md. *Chief, Sec. on Biochem. Pharmacol.* (2, 1954; 3, 1947)

TAFT, EDGAR BRECK, M.D. Massachusetts Gen. Hosp., Boston 14. *Assoc. Pathologist; Asst. Prof. of Pathol., Harvard Med. Sch.* (4, 1956)

TAGER, MORRIS, M.D. Emory U., Dept. of Bacteriol. and Immunol., Atlanta 22, Ga. *Prof. and Chmn. of Dept.* (6, 1948)

TAGGART, JOHN V., M.D. Columbia U., Coll. of P. & S., Dept. of Med., New York City 32. *Prof. of Med.* (1, 1951)

TAHMISIAN, THEODORE N., Ph.D. Argonne Natl. Lab., P. O. Box 299, Lemont, Ill. *Group Leader, Assoc. Biologist.* (1, 1951)

TAINTER, M.L., M.D. Sterling-Winthrop Research Inst., Rensselaer, N.Y. *Dir.* (1, 1929; 3, 1927)

TALALAY, PAUL, M.D. U. of Chicago, Ben May Lab. for Cancer Research, Chicago 37, Ill. *Assoc. Prof.* (2, 1956)

TALBOT, SAMUEL ARMSTRONG, Ph.D. Johns Hopkins Hosp., Baltimore, Md. *Assoc. Prof. of Med., Johns Hopkins Sch. Med.* (1, 1940)

TALIAFERRO, LUCY GRAVES, Sc.D. U. of Chicago, Dept. of Microbiol., Chicago 37, Ill. *Voluntary Res. Assoc.; Assoc. Prof.* (6, 1952)

TALIAFERRO, WILLIAM H., Ph.D. U. of Chicago, Dept. of Microbiol., Chicago 37, Ill. *Eliakim H. Moore Distinguished Serv. Prof. & Chmn.* (6, 1930)

TALLAN, HARRIS H., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (2, 1956)

TALMAGE, DAVID W., M.D. U. of Chicago, Dept. of Med., Chicago 37, Ill. *Assoc. Prof.* (6, 1954)

TALMAGE, ROY V., Ph.D. Rice Inst., Dept. of Biol., Houston, Tex. *Prof. and Chmn.* (1, 1954)

TAMM, IGOR, M.D. Rockefeller Inst., New York City 21. *Assoc. Prof. and Assoc. Physician.* (6, 1954)

TANENBAUM, STUART W., Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Asst. Prof. of Microbiol.* (2, 1957)

TANG, PEI CHIN, Ph.D. USAF Sch. of Aviation Med., Dept. of Physiol.-Biophysics, Randolph AFB, Tex. *Physiologist.* (1, 1955)

TANNENBAUM, ALBERT, M.D. Michael Reese Hosp., 29th and Ellis Ave., Chicago, Ill. *Dir., Dept. of Cancer Research.* (4, 1942)

TARR, H. L. A., Ph.D. Fisheries Research Board, 898 Richards St., Vancouver, Canada. *Dir., Biochem. Dept.* (2, 1950)

TARVER, HAROLD, Ph.D. U. of California Sch. of Med., Dept. of Biochem., 960 Med. Sci. Bldg., San Francisco 22. *Prof.* (2, 1947)

TASAKI, ICHIJI, M.D. Natl. Inst. of Neurol. Diseases and Blindness, NIH, Bethesda 14, Md. *Chief, Special Senses Sec.* (1, 1955)

TASHIRO, SHIRO, Ph.D. M.D. U. of Cincinnati Coll. of Med., Cincinnati, Ohio. *Prof. Emer. of Biol. Chem.* (1R, 1913; 2R, 1913)

TATUM, EDWARD L., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1947)

TAUBER, HENRY, Ph.D. PHS Sch. of Public Health, VD, Exptl. Lab., U. of North Carolina, Chapel Hill. *Assoc. Prof.* (2, 1933)

TAUROG, ALVIN, Ph.D. U. of California Sch. of Med., Berkeley 4. *Assoc. Research Physiologist; Lect. in Physiol.* (2, 1956)

TAYLOR, A. N., Ph.D. U. of Oklahoma Sch. of Med., Oklahoma City 4. *Prof. of Physiol.* (1, 1948)

TAYLOR, ALTON R., Ph.D. Parke, Davis & Co., Research Div., Detroit 32, Mich. *Lab. Dir., Virol.* (6, 1943)

TAYLOR, CHARLES BRUCE, M.D. Presbyterian Hosp., Dept. of Pathol., Chicago, Ill. *Assoc. Prof., U. of Illinois.* (1, 1952; 4, 1952)

TAYLOR, CLARA MAE, Ph.D. Columbia U., Teacher's College, New York City 27. *Prof. of Nutrition.* (5, 1951)

TAYLOR, DERMOT B., M.D., B.Ch., B.A.O. U. of California Med. Ctr., Dept. of Pharmacol., Los Angeles 24. *Prof. and Chmn.* (3, 1950)

TAYLOR, FRED A., Ph.D. 320 E. North Ave., N.S., Pittsburgh, Pa. *Biochemist, Singer Mem. Lab.* (2, 1933)

TAYLOR, HAROLD E., M.D. U. of British Columbia, Faculty of Med., Dept. of Pathol., Vancouver 8, Canada. *Prof. and Head.* (4, 1957)

TAYLOR, HAYWOOD M., Ph.D. Duke U. Sch. of Med., Durham, N.C. *Prof. of Toxicol. and Assoc. Prof. of Biochem.* (4, 1942)

TAYLOR, HENRY LONGSTREET, Ph.D. U. of Minnesota Sch. of Public Health, Gate 27 Stadium, Minneapolis. *Prof. of Physiol. Hygiene.* (1, 1944)

TAYLOR, JOHN FULLER, Ph.D. U. of Louisville Sch. of Med., Dept. of Biochem., Louisville 2, Ky. *Prof. and Chmn.* (2, 1944)

TAYLOR, JULIUS D., Ph.D. Abbott Labs., Dept. of Pharmacol., North Chicago, Ill. *Chem. Pharmacologist.* (3, 1953)

TAYLOR, M. WIGHT, Ph.D. Rutgers, The State U., Dept. of Agric. Biochem., New Brunswick, N. J. *Prof. and Chmn. of Dept.* (5, 1944)

TAYLOR, NORMAN BURKE, M.D. 21 Ardwold Gate, Toronto 10, Canada. (1, 1922)

TAYLOR, REGINALD M., M.D. Columbia U., Coll. of P. & S., Dept. of Psychiatry, New York City 32. *Assoc.; Res. Scientist, Psychiatric Inst.* (1, 1954)

TAYLOR, RICHARD M., M.D., D.P.H. Yale U. Sch. of Med., Sec. of Epidemiol. and Prev. Med., New Haven, Conn. (6, 1949)

TAYLOR, ROBERT D., M.D. Marquette U. Sch. of Med., Milwaukee, Wis. (1, 1945)

TAYLOR, ROBERT E., Ph.D. Natl. Inst. of Neurol. Diseases and Blindness, NIH, Bethesda 14, Md. *Neurophysiologist.* (1, 1957)

TCHEN, TCHE-TSING, Ph.D. Harvard U., Dept. of Chem., 12 Oxford St., Cambridge 38, Mass. *Res. Assoc.* (2, 1958)

TEAGUE, ROBERT S., Ph.D., M.D. U. of Alabama Med. Ctr., Dept. of Pharmacol., Birmingham 3.

Prof. and Chmn. of Dept. (3, 1942)

TEASDALL, ROBERT D., M.D., Ph.D. Johns Hopkins Hosp., Baltimore 5, Md. *Asst. Prof., Neurol. Med.* (1, 1956)

TEDESCHI, DAVID H., Ph.D. Smith Kline & French Labs., Philadelphia 1, Pa. *Sr. Pharmacologist.* (3, 1958)

TEERI, ARTHUR E., Ph.D. U. of New Hampshire, Dept. of Biochem., Durham. *Prof. and Chmn. of Dept.* (5, 1953)

TELFER, WILLIAM H., Ph.D. U. of Pennsylvania, Zool. Lab., Philadelphia. *Asst. Prof.* (6, 1954)

TEMPLETON, ROY D., M.D. 133 Handley St., San Antonio, Tex. (1, 1935)

TEN BROECK, CARL, M.D. Bar Harbor, Maine. 4R, 1932)

TENNENT, DAVID M., Ph.D. Merck Inst. for Therap. Res., Rahway, N.J. *Res. Assoc.* (2, 1958)

TENNEY, STEPHEN MARSH, M.D. Dartmouth Med. Sch., Hanover, N. H. *Prof. of Physiol.* (1, 1956)

TEPLY, LESTER J., Ph.D. 5058 Lake Mendota Dr., Madison, Wis. *Biochemist, Wisconsin Alumni Research Fndn.* (2, 1950; 5, 1954)

TEPPERMAN, HELEN M., Ph.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Asst. Prof. of Pharmacol.* (1, 1955)

TEPPERMAN, JAY, M.D. State U. of New York Upstate Med. Ctr., Syracuse 10. *Prof. of Exptl. Med.* (1, 1944; 3, 1952)

TERNER, CHARLES, Ph.D. Worcester Fndn. for Exptl. Biol., Shrewsbury, Mass. *Assoc. Research Prof. of Biol., Boston U.* (2, 1956)

TERPLAN, KORNEL L., M.D. U. of Buffalo Sch. of Med., Dept. of Pathol., Buffalo, N.Y. *Prof. and Head of Dept.* (4, 1935)

TERRY, ROGER, M.D. U. of Rochester Sch. of Med. and Dent., Dept. of Pathol., Rochester 20, N.Y. (4, 1949)

TERZUOLO, CARLO A., M.D. U. of California, Dept. of Anatomy, Los Angeles 24. *Assoc., Res.* (1, 1958)

THAL, ALAN PHILIP, M.B., Ph.D. U. of Minnesota Hosps., Dept. of Surgery, Minneapolis 14. *Asst. Prof.; Estab. Invest., Amer. Heart Assn.* (4, 1956)

THANNHAUSER, S. J., M.D., Ph.D. Pratt Diagnostic Hosp., 30 Bennet St., Boston, Mass. *Sr. Physician; Prof. of Clin. Med., Tufts U.* (2, 1937)

THATCHER, JONATHAN S., M.D., Ph.D. 750 Kains Ave., San Bruno, Calif. (1, 1949)

THAUER, RUDOLF, M.D. W.G. Kerckhoff-Inst., d. Max-Planck-Gesellschaft, Bad Nauheim, W. Germany. *Dir.; Prof. & Dir., Physiol., U. Giessen.* (1, 1949)

THAYER, SIDNEY ALLEN, Ph.D. St. Louis U. Sch. of Med., St. Louis 22, Mo. *Prof. of Biochem.* (2, 1933)

THERMAN, P. O., M.D. 111 N. 49th St., Philadelphia 39, Pa. *Dir. Res., Inst. of Pa. Hosp.; Asst. Prof., Neurophysiol. Psychiatry, U. of Pa.* (1, 1950)

THESLEFF, STEPHEN, M.D. U. of Lund, Lund, Sweden. (3, 1953)

THIENES, CLINTON H., M.D., Ph.D. Inst. of Med. Research, Huntington Mem. Hosp., Pasadena 2, Calif. *Dir.* (3, 1928)

THOMAS, ARTHUR W., Ph.D. Columbia U., New York City 27. *Prof. of Chem.* (2, 1924)

THOMAS, BYRON H., Ph.D. Iowa State Coll., Ames. *Prof. of Chem., Iowa Agric. Expt. Sta.* (5, 1933)

THOMAS, GARTH J., Ph.D. U. of Illinois, Dept. of Electrical Engin., Urbana. *Res. Prof., Physiol. and Biophysical Res. Lab.* (1, 1958)

THOMAS, J. EARL, M.D. Coll. of Med. Evangelists, Loma Linda, Calif. *Visiting Prof. of Physiol.* (1, 1922; 3, 1924)

THOMAS, LEWIS, M.D. New York U. Coll. of Med., Dept. of Med., New York City 16. *Chmn. of Dept.* (6, 1950)

THOMAS, LLOYD E., Ph.D. Good Samaritan Hosp., Portland 10, Ore. *Consultant in Biochem.* (2, 1951)

THOMAS, WILBUR ADDISON, M.D. Washington U., Dept. of Pathol., St. Louis 10, Mo. *Assoc. Prof.* (4, 1956)

THOMPSON, DAVID D., M.D. Cornell U., Med. Coll., Dept. of Med., New York City 21. *Assoc. Prof.* (1, 1956)

THOMPSON, MARVIN R., Ph.D. 52 Davenport St., Stamford, Conn. *Pres. and Dir. of Research, Marvin R. Thompson, Inc.* (3, 1944)

THOMPSON, RANDALL L., Sc.D., M.D. Sterling-Winthrop Research Inst., Microbiol. Sec., Rensselaer, N.Y. *Head.* (6, 1937)

THOMPSON, RICHARD, M.D. U. of Colorado, Denver 20. *Prof. of Microbiol.* (6, 1951)

THOMPSON, WILLIAM R., Ph.D. 1 Darroch Rd., Delmar, N.Y. *Sr. Biochemist, Div. of Labs. and Res., N. Y. State Dept. of Health.* (2, 1934)

THOMSON, DAVID LANDSBOROUGH, Ph.D., D.Sc. McGill U., Montreal, Canada. *Prof. of Biochem. and Dean, Faculty of Grad. Studies.* (2, 1929)

THOMSON, JOHN D., Ph.D. State U. of Iowa, 240 Med. Labs., Iowa City. *Assoc. Prof. of Physiol.* (1, 1951)

THOMSON, JOHN F., Ph.D. Argonne Natl. Lab., Div. of Biol. and Med. Res., Lemont, Ill. *Assoc. Pharmacologist.* (1, 1958)

THORN, GEORGE WIDMER, M.D. Peter Bent Brigham Hosp. Boston, Mass. *Physician-in-Chief; Prof. of Med., Harvard U.* (1, 1939)

THORNE, CURTIS B., Ph.D. Fort Detrick, MB Div., Frederick, Md. *Chief, Microbial Physiol. Br.* (2, 1955)

THORP, W. T. S., D.V.M., M.S. U. of Minnesota Coll. of Veterinary Med., St. Paul 1. *Dean.* (4, 1948)

TIDWELL, HERBERT C., Ph.D. U. of Texas Southwestern Med. Sch., Dept. of Biochem., Dallas. *Prof. and Chmn. of Dept.* (2, 1948; 5, 1955)

TIGERTT, WILLIAM DAVID, M.D. Walter Reed Army Inst. of Research, Washington 12, D.C. *Dir., Div. Spec. Operations.* (6, 1956)

TILLETT, WILLIAM S., M.D. New York U., Coll. of Med., Dept. of Med., New York City 16. *Emer. Prof.* (6, 1927)

TILT, JENNIE, Ph.D. 1011 Lincoln Blvd., Santa Monica, Calif. (5R, 1939)

TIMIRAS, PAOLA S., M.D., Ph.D. U. of California Sch. of Med., Dept. of Physiol., Berkeley 4. (3, 1952)

TIPSON, R. STUART, Ph.D., D.Sc. Natl. Bureau of Standards, Washington 25, D.C. *Organic Chemist.* (2, 1937)

TIPTON, SAMUEL R., Ph.D. U. of Tennessee, Dept.

of Zool., Knoxville. *Prof.* (1, 1940)

TISLOW, RICHARD F., M.D., Dr. Med. Sci. Wyeth Inst. for Med. Res., P.O. Box 8299, Philadelphia 1, Pa. *Chief Pharmacologist.* (1, 1944; 3, 1950)

TITUS, ELWOOD O., Ph.D. Natl. Heart Inst., Lab. of Chem. Pharmacol., NIH, Bethesda 14, Md. *Chief, Sec. on Organic Chem.* (2, 1957; 3, 1958)

TITUS, HARRY W., Ph.D. Lime Crest Research Lab., R.D. 1, Box 67, Newton, N.J. *Tech. Counsellor and Dir. of Research.* (2, 1929; 5, 1933)

TOBIAN, LOUIS, JR., M.D. U. of Minnesota Sch. of Med., Minneapolis 14. *Assoc. Prof. of Med.* (1, 1955)

TOBIAS, JULIAN M., M.D., Ph.D. U. of Chicago, Chicago 37, Ill. *Prof. of Physiol.* (1, 1944)

TOCANTINS, LEANDRO MAUES, M.D. Jefferson Med. Coll., Philadelphia, Pa. *Prof. of Clin. and Exptl. Med.* (1, 1939)

TODD, WILBERT R., Ph.D. U. of Oregon Med. Sch., Portland. *Prof. of Biochem.* (2, 1948; 5, 1948)

TODHUNTER, ELIZABETH NEIGE, Ph.D. U. of Alabama School of Home Econ., University. *Dean.* (5, 1939)

TOENNIES, GERRIT, Ph.D. Inst. for Cancer Research, Dept. of Gen. Biochem., Philadelphia 11, Pa. *Head of Dept.* (2, 1934)

TOLBERT, BERT M., Ph.D. U. of Colorado, Chem. Dept., Boulder. *Assoc. Prof.* (2, 1958)

TOLBERT, N. EDWARD, Ph.D. Michigan State U., Dept. of Agric. Chem., East Lansing. *Prof.* (2, 1953)

TOLLE, CHESTER D., Ph.D. Food and Drug Admin., Dept. of HEW, Washington 25, D.C. *Asst. Dir., Div. of Nutrition.* (5, 1942)

TOMAN, JAMES E. P., Ph.D. Chicago Med. Sch., Dept. of Physiol. and Pharmacol., Chicago, Ill. *Assoc. Prof.* (1, 1945; 3, 1950)

TOMARELLI, RUDOLPH M., Ph.D. Wyeth Inst. for Med. Research, Radnor, Pa. *Sr. Invest.* (2, 1950; 5, 1950)

TOMASHEFSKI, JOSEPH F., M.D. Ohio State U., Columbus 10. *Asst. Prof. of Med. and Physiol.; Chief of Res., Obio Tuberculosis Hosp.* (1, 1956)

TOMKINS, GORDON M., M.D., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Sr. Surgeon, PHS.* (2, 1957)

TOMLINSON, WRAY JOSEPH, M.D. The Medical Ctr., Columbus, Ga. (4, 1945)

TOMPKINS, EDNA H., M.D. New England Deaconess Hosp., Cancer Research Inst. Boston 15, Mass. *Research Assoc.* (4, 1941)

TOMPKINS, VICTOR N., M.D. New York State Health Dept., Div. of Labs. and Res., Albany 1. *Dir.; Assoc. Prof. Pathol., Albany Med. Coll.* (4, 1957)

TOOLAN, HELENE WALLACE, Ph.D. Sloan-Kettering Inst., New York City 21. *Assoc.; Assoc. Prof. of Pathol., Cornell U.* (4, 1956)

TOPPER, YALE J., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. (2, 1952)

TOTH, LOUIS A., Ph.D. Louisiana State U. Sch. of Med., Dept. of Physiol., New Orleans 13. *Prof.* (1, 1940)

TOTTER, JOHN R., Ph.D. U. of Montevideo, Montevideo, Uruguay. (2, 1946)

TOUCHSTONE, JOSEPH C., Ph.D. Hosp. of U. of Pennsylvania, Philadelphia 4. *Asst. Prof., Dept. Obstetrics & Gynecol., U. of Pa.* (2, 1958)

TOURTELLOTTE, DEE, D.Sc. Charles B. Knox Gelatine Co., 5th and Erie Sts., Camden 2, N.J. *Res. Dir.* (5, 1935)

TOUSTER, OSCAR, Ph.D. Vanderbilt U. Sch. of Med., Dept. of Biochem. Nashville, Tenn. *Prof.* (2, 1952)

TOWE, ARNOLD L., Ph.D. U. of Washington Sch. of Med., Dept. of Physiol. and Biophysics, Seattle 5. *Asst. Prof.* (1, 1958)

TOWER, SARAH SHELDON, M.D., Ph.D. 11 E. Chase St., Baltimore 2, Md. *Asst. of Psychiatry, Johns Hopkins U., Sch. Med.* (1, 1932)

TOWNSEND, FRANK M., M.D. Armed Forces Inst. of Pathol., Washington 12, D.C. *Dep. Dir.* (4, 1958)

TRAGER, WILLIAM, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Assoc. Prof.* (4, 1947)

TRAPOLD, JOSEPH H., Ph.D. Louisiana State U. Sch. of Med., Dept. of Pharmacol., New Orleans. *Assoc. Prof.* (3, 1955)

TRAUB, FREDERICK B., M.D. 405 E. 72nd St., New York City 21. *Microbiologist, Jewish Hosp. of Brooklyn.* (6, 1946)

TRAVELL, JANET, M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Clin. Pharmacol.* (3, 1933)

TRAVIS, LEE EDWARD, Ph.D. Gibraltar Savings & Loan Assn., Beverly Hills, Calif. *Dir. and Vice Pres.* (1, 1929)

TREADWELL, CARLETON R., Ph.D. George Washington U., Sch. of Med., Dept. of Biochem., Washington 5, D.C. *Prof.* (2, 1944; 5, 1949)

TREFFERS, HENRY P., Ph.D. Yale U. Sch. of Med., Dept. of Microbiol., New Haven 11, Conn. *Prof.* (6, 1942)

TRENDELENBURG, ULLRICH GEORG, M.D., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. in Pharmacol.* (3, 1958)

TRIMBLE, HARRY C., Ph.D., Sc.D. Harvard Med. Sch., Boston 15, Mass. *Spec. Consultant to the Dean.* (2, 1929; 5R, 1936)

TROLL, WALTER, Ph.D. New York U., Coll. of Med., Inst. of Indust. Med., New York City 16. *Asst. Prof.* (2, 1957)

TRUANT, ALDO P., Ph.D. Astra Pharmaceut. Products, Inc., Worcester 6, Mass. *Dir., Biol. Res.; Lect., Pharmacol., Tufts U.* (3, 1955)

TRUCCO, RAUL E., Ph.D. Oklahoma Med. Research Fndn., Psychosomatic and Neuromuscular Sec., Oklahoma City 4. *Assoc. Head.* (2, 1957)

TRUITT, EDWARD B., JR., Ph.D. U. of Maryland Sch. of Med., Dept. of Pharmacol., Baltimore 1. (3, 1955)

TRULSON, MARTHA F., D.Sc. Harvard Sch. of Public Health, Boston 15, Mass. *Assoc. Prof. of Nutrition.* (5, 1956)

TRURNIT, HANS JOACHIM, M.D. Research Inst. for Advanced Study, Baltimore 12, Md. *Sr. Scientist.* (1, 1950)

TSCHIRGI, ROBERT D., M.D., Ph.D. U. of California Sch. of Med., Depts. of Physiol. and Anatomy, Los Angeles 24. *Assoc. Prof.* (1, 1951)

TSUJI, FREDERICK I., Ph.D. VA Hosp., Pittsburgh 40, Pa. *Tech. Dir., Research Lab.* (2, 1956)

TUBA, JULES, Ph.D. U. of Alberta, Dept. of Biochem., Edmonton, Canada. *Prof.* (2, 1950)

TUCKER, ROBERT G., Ph.D. Smith Kline & French Labs., Res. & Develop. Div., Philadelphia 1, Pa. *Group Leader in Gen. Biol. Res.* (5, 1958)

TUFT, LOUIS, M.D. 1530 Locust St., Philadelphia, Pa. *Clin. Prof. Med., Temple U.; Chief, Clinic of Allergy & Appl. Immunol., Temple Hosp.* (6, 1928)

TULLIS, JOHN LOUIS, M.D. New England Deaconess Hosp., Cancer Research Inst., Boston 15, Mass. *Pathologist.* (4, 1952)

TUM SUDEN, CAROLINE, Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Pharmacologist, Pharmacol. Br.* (1, 1936)

TUNTURI, ARCHIE ROBERT, Ph.D., M.D. U. of Oregon Med. Sch., Portland 1. *Asst. Prof. of Anatomy.* (1, 1946)

TURNER, ROBERT A., Ph.D. Warner-Chilcott Labs., Morris Plains, N. J. (2, 1952)

TUTTLE, WAID WRIGHT, Ph.D., Sc.D. State U. of Iowa, Iowa City. *Prof. of Physiol.* (1, 1925)

TYLER, ALBERT, Ph.D. California Inst. of Technology, Pasadena, Calif. *Prof. of Embryol.* (6, 1946)

TYLER, DAVID B., Ph.D. U. of Puerto Rico Schools of Dent. and Med., Dept. of Pharmacol., San Juan 22. *Prof. and Dir.* (1, 1943; 3, 1951)

TYTELL, ALFRED A., Ph.D. Merck Inst. for Therapuetic Res., Cell Biol. Dept., West Point, Pa. *Dir.* (2, 1951; 6, 1950)

UDENFRIEND, SIDNEY, Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Clin. Biochem.* (2, 1950; 3, 1956)

UHR, JONATHAN W., M.D. New York U. Coll. of Med., Dept. of Med., New York City 16. *Asst. Prof.; Sr. Research Fellow, PHS.* (6, 1957)

UMBARGER, H. EDWIN, Ph.D. Harvard Med. Sch., Dept. of Bacteriol. and Immunol., Boston 15, Mass. *Asst. Prof.* (2, 1954)

UMBREIT, WAYNE W., Ph.D. Rutgers, The State U., Dept. of Bacteriol., New Brunswick, N. J. *Head of Dept.* (2, 1947)

UNDERKOFLER, LELAND A., Ph.D. Takamine Lab., Div. of Miles Labs., Inc., Clifton, N. J. *Research Dir.* (2, 1953)

UNGAR, GEORGES, M.D., D.Sc. U. S. Vitamin Corp., Dept. of Pharmacol., Yonkers, N. Y. *Dir.* (1, 1950)

UNGER, LESTER J., M.D. New York U.-Bellevue Med. Ctr., New York City 28. *Dir., Blood and Plasma Bank.* (6, 1952)

UNGLAUB, WALTER G., M.D. Tulane U., Sch. of Med., New Orleans 12, La. *Assoc. Prof. of Med.* (5, 1956)

UNNA, KLAUS R. W., M.D. U. of Illinois Coll. of Med., Dept. of Pharmacol., Chicago 12. *Prof. and Head of Dept.* (1, 1941; 3, 1944; 5, 1942)

UPTON, ARTHUR C., M.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Pathologist.* (4, 1954)

UPTON, MORGAN, Ph.D. Rutgers, The State U., Dept. of Psychol. New Brunswick, N. J. (1, 1934)

URSILLO, RICHARD C., Ph.D. U. of California Med. Ctr., Los Angeles 24. *Asst. Prof. of Pharmacol.* (3, 1957)

UTTER, MERTON F., Ph.D. Western Reserve U., Dept. of Biochem., Cleveland, Ohio. *Prof.* (2, 1946)

VAHLTEICH, ELLA McCOLLUM, Ph.D. 310 Walnut St., Englewood, N. J. *Nutrition Specialist.* (5, 1933)

VALLE, J. R., M.D. Escola Paulista de Medicina, Caixa Postal 12993, São Paulo, Brazil. *Prof. of Pharmacol.* (3, 1947)

VALLEE, BERT L., M.D. Peter Bent Brigham Hosp., Harvard Med. Sch., Biophysics Research Lab., Boston 15, Mass. *Scientific Dir.* (2, 1956)

VAN ARMAN, C. G., Ph.D. G. D. Searle & Co., Box 5110, Chicago 80, Ill. *Pharmacologist.* (3, 1953)

VANATTA, JOHN C., M.D. U. of Texas Southwestern Med. Sch., Dept. of Physiol., Dallas. *Prof.* (1, 1955)

VAN BRUGGEN, JOHN T., Ph.D. U. of Oregon Med. Sch., Portland. *Assoc. Prof. of Biochem.* (2, 1952)

VANDER BROOK, MILTON J., Ph.D. The Upjohn Co., Dept. of Pharmacol., Kalamazoo 99, Mich. *Head.* (3, 1950)

VAN DYKE, H. B., Ph.D., M.D. Columbia U., Coll. of P. & S., New York City 32. *David Hosack Prof. of Pharmacol.* (1, 1925; 3, 1927)

VAN HARREVELD, ANTHONIE, M.D. California Inst. of Technology, Pasadena. *Prof. of Physiol.* (1, 1941)

VAN ITALLIE, THEODORE B., M.D. St Luke's Hosp., New York City 25. *Dir. of Med.* (5, 1956)

VAN LANCKER, JULIEN L., M.D. U. of Utah Coll. of Med., Salt Lake City. *Asst. Prof. of Patbol.* (4, 1957)

VAN LIERE, EDWARD J., M.D., Ph.D. West Virginia U. Sch. of Med., Morgantown. *Prof. of Physiol. and Dean.* (1, 1927)

VAN MAANEN, EVERT FLORUS, Ph.D., The Wm. S. Merrell Co., Cincinnati 15, Ohio. *Assoc. Dir. of Research in Charge of Biol. Sci.* (3, 1951)

VAN MIDDLESWORTH, LESTER, Ph.D., M.D. U. of Tennessee, Memphis. *Assoc. Prof. of Physiol.; Instr. of Med.* (1, 1948)

VAN PILSUM, JOHN F., Ph.D. U. of Minnesota, Dept. of Physiol. Chem., Minneapolis 14. *Asst. Prof.* (2, 1958)

VAN REEN, ROBERT, Ph.D. Naval Med. Research Inst., Natl. Naval Med. Ctr., Bethesda 14, Md. *Biochemist.* (5, 1956)

VAN SLYKE, DONALD D., Ph.D., M.D. Brookhaven Natl. Lab., Med. Dept., Upton, N. Y. *Chemist; Member Emer., Rockefeller Inst.* (2, 1908)

VAN VUNAKIS, HELEN, Ph.D. New York State Dept. of Health, Div. of Labs. & Res., Albany 1. *Sr. Res. Scientist.* (2, 1958)

VAN WAGENEN, GERTRUDE, Ph.D. Yale U. Sch. of Med., New Haven 11, Conn. *Assoc. Prof.* (1, 1932)

VAN WAGTENDONK, WILLEM J., Ph.D. Indiana U., Dept. of Zool., Bloomington. *Prof.* (2, 1946)

VAN WINKLE, WALTON, JR., M.D. Ethicon, Inc., Research Div., Somerville, N. J. *Vice Pres., Research.* (3, 1939)

VARGA, LASZLO, Ph.D. 326 Main St., Winchester, Mass. *Assoc. Member, Retina Fndn., Boston.* (2, 1957)

VARNER, J. E., Ph.D. Ohio State U., Dept. of Agric.

Biochem., Columbus 10. *Assoc. Prof.* (2, 1958)

VARNEY, PHILIP L., Ph.D. Biological Research, Inc., 5373 Bermuda, St. Louis 21, Mo. *Bacteriologist, St. Luke's Hosp.* (6, 1948)

VARS, HARRY M., Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Prof. of Biochem. in Surgical Res.* (2, 1935; 5, 1935)

VAUGHAN, JOHN H., M.D. U. of Rochester Sch. of Med. and Dent., Rochester 20, N.Y. (6, 1954)

VAWTER, GORDON FULLER, M.D. Children's Hosp., Boston, Mass. *Asst. Patbologist.* (4, 1956)

VAZQUEZ, JACINTO J., M.D. U. of Pittsburgh Sch. of Med., Pittsburgh 13, Pa. *Asst. Prof. of Patbol.* (4, 1958)

VELARDO, JOSEPH THOMAS, Ph.D. Yale U., Sch. of Med., Dept. of Anatomy, New Haven 11, Conn. *Asst. Prof.* (1, 1954)

VELICK, SIDNEY FREDERICK, Ph.D. Washington U., Sch. of Med., Dept. of Biochem., St. Louis 10, Mo. *Assoc. Prof.* (2, 1946)

VENNESLAND, BIRGIT, Ph.D. U. of Chicago, Dept. of Biochem., Chicago 37, Ill. *Prof.* (2, 1944)

VENNING, ELEANOR H., Ph.D. University Clinic, Royal Victoria Hosp., Montreal, Canada. *Assoc. Prof. Exptl. Med., McGill U.* (2, 1938)

VERNIER, ROBERT L., M.D. U. of Minnesota Sch. of Med., Dept. of Pediatrics, Minneapolis 14. *Amer. Heart Assn. Res. Fellow.* (4, 1957)

VERNIER, VERNON G., M.D. Merck Inst. for Therap. Research, Merck & Co., Inc., West Point, Pa. *Research Assoc., Physiol. Dept.* (3, 1957)

VERNON, LEO P., Ph.D. Brigham Young U., Chem. Dept., Provo, Utah. *Assoc. Prof.* (2, 1955)

VERZEANO, MARCEL, M.D. U. of California Sch. of Med., Dept. of Biophysics, Los Angeles 24. *Assoc. Prof.* (1, 1955)

VESTLING, CARL SWENSSON, Ph.D. U. of Illinois, Biochem. Div., Noyes Lab., Urbana. *Prof.* (2, 1946)

VICK, ROBERT L., Ph.D. U. of Cincinnati Coll. of Med., Dept. of Pharmacol., Cincinnati 19, Ohio. *Res. Assoc.* (3, 1958)

VICKERY, HUBERT B., Ph.D. Connecticut Agric. Expt. Sta., New Haven. *Chief Biochemist; Lect., Yale U.* (2, 1923)

VILLARREAL, RAMON, M.D. Escuela de Medicina Univ. de San Luis Potosi, San Luis Potosi, Mexico. *Prof. and Head, Physiol.* (1, 1955)

VILLEE, CLAUDE A., JR., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Assoc. Prof. of Biol. Chem.* (2, 1948)

VIRTUE, ROBERT W., Ph.D., M.D. U. of Colorado Med. Ctr., Denver 20. *Prof. of Anesthesiol.* (2, 1939)

VISHNIAC, WOLF, Ph.D. Yale U., Dept. of Microbiol., New Haven 11, Conn. *Assoc. Prof.* (2, 1954)

VISSCHER, FRANK E., Ph.D. Health Specialties, Inc., 343 N. Rose St., Kalamazoo, Mich. (1, 1947)

VISSCHER, MAURICE B., Ph.D., M.D. U. of Minnesota, Dept. of Physiol., Minneapolis. *Prof. and Head of Dept.* (1, 1927)

VISSER, DONALD W., Ph.D. U. of Southern California, Los Angeles 7. *Prof. of Biochem.* (2, 1952)

VITALE, JOSEPH J., D.Sc. Harvard Sch. of Public Health, Boston 15, Mass. *Asst. Prof. of Nutrition.* (5, 1958)

VOEGTLIN, CARL, Ph.D. 4700 Connecticut Ave., N.W., Washington 8, D.C. (1R, 1908; 2R, 1908; 3R, 1908)

VOGEL, F. STEPHEN, M.D. Cornell U., Med. Coll., New York City 21. *Assoc. Prof. of Patbol.* (4, 1951)

VOGEL, HENRY J., Ph.D. Rutgers, The State U., Inst. of Microbiol., New Brunswick, N.J. *Assoc. Prof. of Microbial Biochem.* (2, 1958)

VOGEL, PETER, M.D. 51 East 90th St., New York City 28. (6, 1951)

VOLK, BRUNO W., M.D. Jewish Chronic Disease Hosp., Isaac Albert Research Inst., Brooklyn 3, N.Y. *Dir.* (4, 1954)

VOLKIN, ELLIOT, Ph.D. Oak Ridge Natl. Lab., Biol. Div., Oak Ridge, Tenn. *Sr. Biochemist.* (2, 1953)

VON FRISCH, KARL. Zool. Inst., U. of Munich, Munich, Germany. (1H, 1952)

VON HAAM, EMMERICH, M.D. Ohio State U., Columbus. *Prof. of Patbol.* (4, 1938)

VON KAULLA, KURT N., M.D. U. of Colorado Sch. of Med., Denver 20. *Asst. Prof. of Med. (Research).* (1, 1957)

VON KORFF, RICHARD W., Ph.D. U. of Minnesota, Dept. of Pediatrics, Room 423a Heart Hosp., Minneapolis 14. *Asst. Prof.* (2, 1954)

VON OETTINGEN, W. F., M.D., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Med. Officer, Toxicol.* (3, 1925)

VORIS, LEROY, Ph.D. Natl. Research Council, 2101 Constitution Ave., Washington 25, D.C. *Exec. Sec., Food and Nutrition Board.* (5, 1949)

VORWALD, ARTHUR J., Ph.D., M.D. Wayne State U. Coll. of Med., Detroit 7, Mich. *Prof. of Indust. Med.* (4, 1937)

VOS, BERT J., Ph.D., M.D. Food and Drug Admin., Div. of Pharmacol., Dept. of HEW, Washington 25, D.C. *Asst. Chief.* (3, 1941)

WACHSTEIN, MAX, M.D. St. Catherine's Hosp., Brooklyn, N.Y. *Dir. of Lab.* (4, 1947)

WADDELL, JAMES, Ph.D. E. I. du Pont de Nemours and Co., Grasselli Chem. Dept., Wilmington 98, Del. *Mgr., Tech. Develop., Nutrition Sec.* (2, 1930; 5, 1935)

WADDELL, WILLIAM JOSEPH, M.D. U. of North Carolina, Dept. of Pharmacol., Chapel Hill. *PHS Res. Fellow.* (3, 1958)

WADE, NELSON, J., Ph.D. St. Louis U., St. Louis 4, Mo. *Prof. of Biol.* (1, 1956)

WAELSCH, HEINRICH, M.D., Sc.D. New York State Psychiatric Inst., New York City 32. *Prof. of Biochem., Columbia U.* (2, 1941)

WAGMAN, IRVING H., Ph.D. Mt. Sinai Hosp., Dept. of Neurol., New York City 29. (1, 1946)

WAGNER, BERNARD M., M.D. U. of Washington Sch. of Med., Dept. of Pathol., Seattle 5. *Assoc. Prof.* (4, 1956)

WAGNER, HENRY G., M.D. Naval Med. Research Inst., Bethesda 14, Md. *Capt. MC, USN.* (1, 1955)

WAGNER, ROBERT R., M.D. Johns Hopkins U., Dept. of Med., Baltimore 5, Md. *Assoc. Prof. of Med.; Physician.* (6, 1953)

WAGNER-JAUREGG, THEODOR, Ph.D. Siegfried A.

G., Zofingen, Switzerland. *Chief, Research Dept.* (2, 1951)

WAINIO, WALTER W., Ph.D. Rutgers, The State U., Bur. of Biol. Res., Box 515, New Brunswick, N. J. *Assoc. Prof. of Biochem.* (2, 1950)

WAISMAN, HARRY A., M.D., Ph.D. U. of Wisconsin Med. Sch., Dept. of Pediatrics, Madison 6. *Prof.* (2, 1944)

WAKERLIN, GEORGE E., Ph.D., M.D. Amer. Heart Assn., 44 E. 23rd St., New York City 10. *Med. Dir.* (1, 1933; 3, 1934)

WAKIL, SALIH J., Ph.D. U. of Wisconsin, Inst. for Enzyme Res., Madison 5. *Asst. Prof. of Enzyme Chem.* (2, 1958)

WAKIM, KHALIL G., M.D., Ph.D. Mayo Clinic, Rochester, Minn. *Consultant; Prof. of Physiol. Mayo Fndn.* (1, 1942; 3, 1951)

WAKSMAN, BYRON H., M.D. Harvard U. Med. Sch., Boston 15, Mass. *Asst. Prof. in Bacteriol. and Immunol.* (6, 1950)

WALCOTT, WILLIAM W., Ph.D. Columbia U., Coll. of P. & S., Dept. of Physiol., New York City 32. *Assoc. Prof.* (1, 1947)

WALD, GEORGE, Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Prof. of Biol.* (2, 1950)

WALDRON, JEROME M., M.D. Temple U. Sch. of Med., Dept. of Physiol., Philadelphia 40, Pa. *Assoc. Prof.* (1, 1952)

WALFORD, ROY LEE, JR., M.D. U. of California Sch. of Med., Los Angeles. *Asst. Prof. of Pathol.* (4, 1956)

WALKER, A. EARL, M.D. 601 N. Broadway, Baltimore 5, Md. *Prof. of Neurol. Surgery, Johns Hopkins U.* (1, 1950)

WALKER, BURNHAM S., Ph.D., M.D. Burbank Hosp., Fitchburg, Mass. (2, 1940)

WALKER, DUARD L., M.D. U. of Wisconsin Med. Sch., Madison 6. *Assoc. Prof. of Med. Microbiol.* (6, 1958)

WALKER, JAMES B., Ph.D. Baylor U. Coll. of Med., Houston, Tex. *Asst. Prof. of Biochem.* (2, 1955)

WALKER, SHEPPARD M., Ph.D. U. of Louisville Sch. of Med., Louisville, Ky. *Assoc. Prof. of Physiol.* (1, 1946)

WALL, PATRICK DAVID, M.A., B.M., B.Ch. Massachusetts Inst. of Technology, 16-509, Cambridge. *Assoc. Prof., Biol.* (1, 1953)

WALLACE, HAROLD D., Ph.D. Agric. Expt. Sta., U. of Florida, Gainesville. *Assoc. Prof. of Animal Husbandry and Nutrition.* (5, 1958)

WALLACE, JOHN H., Ph.D. Harvard Med. Sch., Dept. Bacteriol. & Immunol., Boston 15, Mass. *Res. Assoc.; Asst. Bacteriol., Wood Mem.* (6, 1958)

WALLEN-LAWRENCE, ZONJA, Ph.D. 4534 W. Pine Blvd., St. Louis 8, Mo. (2, 1937)

WALSER, MACKENZIE, M.D. Johns Hopkins U., Sch. of Med., Baltimore 5, Md. *Asst. Prof. of Pharmacol. & Exptl. Therap.; Asst. Prof. of Med.* (1, 1957)

WALTER, ANNABEL W. 86-42 251st St., Bellerose 6, N. Y. *Bacteriologist.* (6, 1946)

WALTER, CARL W., M.D. 721 Huntington Ave., Boston 15, Mass. *Assoc. Clin. Prof. of Surgery, Harvard U.; Surgeon, P. B. Brigham Hosp.* (4, 1942)

WALTERS, ORVILLE S., Ph.D., M.D. U. of Illinois, Champaign. *Dir. of Health Serv.; Prof. of Hygiene; Lect. in Psychiatry.* (1, 1936)

WALTON, ROBERT P., Ph.D., M.D. Med. Coll. of South Carolina, Charleston 16. *Prof. of Pharmacol.* (3, 1933)

WALTON, SETH T., V.M.D., Ph.D. State of North Carolina Lab. of Hygiene, Raleigh. *Bacteriologist.* (6, 1936)

WALZER, MATTHEW, M.D. 20 Plaza St., Brooklyn, N. Y. *Consultant in Allergy, Jewish Hosp. of Brooklyn.* (6, 1924)

WANG, CHI CHE, Ph.D. Winter VA Hosp., Topeka, Kans. *Biochemist.* (2, 1922; 5, 1933)

WANG, CHIH HSING, Ph.D. Oregon State Coll., Dept. of Chem., Corvallis. *Prof.* (2, 1955)

WANG, G. H., Ph.D. U. of Wisconsin Sch. of Med., Madison 6. *Project Assoc.* (1, 1956)

WANG, RICHARD I. H., M.D., Ph.D. 2900 Galiano St., Coral Gables, Fla. (3, 1957)

WANG, SHIH-CHUN, M.D., Ph.D. Columbia U., Coll. of P. & S., Dept. of Pharmacol., New York City 32. *Prof.* (1, 1943; 3, 1952)

WANG, TUNG-YUE, Ph.D. Washington U., Botany Dept., St. Louis 5, Mo. *Asst. Prof.* (2, 1957)

WANGEMAN, CLAYTON P., M.D. Associated Anesthesiologists, 1110 Harvard Ave., Seattle 22, Wash. (3, 1946)

WANGENSTEEN, OWEN H., M.D. University Hosp., Minneapolis 14, Minn. *Prof. of Surgery, U. of Minnesota.* (1, 1947; 4, 1931)

WARD, ARTHUR A., JR., M.D. U. of Washington Sch. of Med., Div. of Neurosurgery, Seattle. *Head; Prof. Surgery Neurosurgery.* (1, 1949)

WARD, ROBERT, M.D. U. of Southern California, Dept. of Pediatrics, Children's Hosp., 4614 Sunset Blvd., Los Angeles, Calif. (6, 1951)

WARD, THOMAS G., M.D., Dr.P.H. U. of Notre Dame, Notre Dame, Ind. *Prof. of Virol.* (6, 1958)

WARD, WALTER E., Ph.D., M.D. Cutter Labs., Berkeley 10, Calif. *Med. Dir.* (6, 1947)

WARE, ARNOLD G., Ph.D. Los Angeles County Hosp., Main Lab., Los Angeles 33, Calif. *Head Chemist; Prof., Dept. Biochem., U. of Southern Calif.* (2, 1949)

WARNER, EMORY D., M.D. State U. of Iowa, Dept. of Pathol., Med. Labs. Bldg., Iowa City. *Prof. and Head of Dept.* (4, 1937)

WARNER, GEORGE F., M.D. Orange County Gen. Hosp., Orange, Calif. *Chief of Clin. Serv.* (1, 1956)

WARNER, HOMER R., M.D., Ph.D. Latter Day Saints Hosp., Cardiovasc. Lab., Salt Lake City, Utah. *Dir.; Asst. Res. Prof. Physiol., U. of Utah.* (1, 1957)

WARNER, ROBERT C., Ph.D. New York U. Coll. of Med., New York City 16. *Assoc. Prof. of Biochem.* (2, 1946)

WARREN, JAMES V., M.D. Duke U. Sch. of Med., Dept. of Med., Durham, N. C. *Prof.* (1, 1947)

WARREN, JOEL, Ph.D. Charles Pfizer & Co., Inc., Terre Haute, Ind. *Dir.; Biologics Research.* (6, 1949)

WARREN, MARSHALL R., Ph.D. 6901 Old State Rd., Evansville 10, Ind. (3, 1948)

WARREN, SHIELDS, M.D. 194 Pilgrim Rd., Boston 15, Mass. *Prof. of Pathol., Harvard Med. Sch.* (4, 1929)

WARTMAN, WILLIAM BECHMANN, M.D. Northwestern U., Chicago 11, Ill. *Morrison Prof. of Pathol.* (4, 1940)

WASSERMAN, KARLMAN, M.D., Ph.D. Johns Hopkins Hosp., Baltimore 5, Md. (1, 1954)

WASSERMAN, ROBERT H., Ph.D. New York State Veterinary Coll., Lab. of Radiation Biol., Cornell U., Ithaca. *Res. Assoc.* (5, 1958)

WASTENEYS, HARDOLPH, Ph.D. U. of Toronto, Toronto, Canada. *Prof. Emer. of Biochem.* (2R, 1915)

WATERMAN, ROBERT E., B.S. Schering Corp., 60 Orange St., Bloomfield, N. J. *Vice-Pres.* (2, 1940)

WATERS, RALPH MILTON, M.D. R.R. #3, Box 433, Orlando, Fla. (3R, 1937)

WATKIN, DONALD MORGAN, M.D. Natl. Cancer Inst., Nutrition and Metabolism Serv., NIH, Bethesda 14, Md. *Sr. Invest.* (1, 1957; 5, 1958)

WATSON, BARBARA K., Ph.D. Harvard Med. Sch., Boston 15, Mass. *Res. Assoc. in Bacteriol. and Immunol.* (6, 1953)

WATSON, CECIL J., M.D. University Hosp., Dept. of Med., Minneapolis, Minn. *Prof.* (2, 1952)

WATSON, DENNIS W., Ph.D. U. of Minnesota Med. Sch., Dept. of Bacteriol. and Immunol., Minneapolis. *Prof.* (6, 1949)

WATSON, JAMES D., Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Asst. Prof. of Biol.* (2, 1958)

WATSON, JOHN B., Ph.D. Park Road, Woodbury, Conn. (1, 1907)

WATTENBERG, LEE W., M.D. U. of Minnesota, Dept. of Pathol., Minneapolis 14. *Asst. Prof. (Lederle Med. Faculty Awardee).* (4, 1957)

WATTS, DANIEL T., Ph.D. West Virginia U. Med. Ctr., Dept. of Pharmacol., Morgantown. *Prof. and Head of Dept.* (3, 1950)

WAUD, RUSSELL A., M.D., Ph.D. U. of Western Ontario Med. Sch., Dept. of Pharmacol., London, Canada. *Prof. and Head of Dept.* (3, 1931)

WAUGH, DAVID F., Ph.D. Massachusetts Inst. of Technology, Dept. of Biol., Cambridge. *Prof. of Biophysics.* (1, 1943; 2, 1956)

WAY, E. LEONG, Ph.D. U. of California Med. Ctr., San Francisco 22. *Prof. of Pharmacol. and Toxicol.* (3, 1947)

WAY, JAMES L., Ph.D. U. of Wisconsin, Serv. Mem. Inst., Dept. of Pharmacol., Madison 6. *Instr.* (3, 1957)

WEARN, JOSEPH T., M.D. University Hosps. of Cleveland, Cleveland 6, Ohio. *Dean, Western Reserve U., Sch. of Med.* (1, 1921)

WEATHERBY, J. H., Ph.D. Med. Coll. of Virginia, Dept. of Physiol. and Pharmacol., Richmond 19. *Assoc. Prof. of Pharmacol.* (3, 1941)

WEAVER, LAWRENCE C., Ph.D. Pitman-Moore Co., Indianapolis 6, Ind. *Pharmacologist.* (3, 1955)

WEBB, PAUL, M.D. Aero Med. Lab., Wright Air Develop. Ctr., Wright-Patterson AFB, Ohio. *Med. Officer, Aviation Med.* (1, 1956)

WEBER, CLARENCE J., M.D., Ph.D. 156 Overhill Rd., Salina, Kans. (2, 1931)

WEBSTER, BRUCE, M.D., C.M. Cornell U., Med. Coll., New York City 21. *Assoc. Prof. of Med.; Assoc. Attending Physician, New York Hosp.* (5, 1935)

WEBSTER, GEORGE C., Ph.D. Ohio State U., Dept. of Agric. Biochem., Columbus 10. *Assoc. Prof.* (2, 1955)

WEDGWOOD, RALPH J. P., M.D. Babies and Children's Hosp., Cleveland 6, Ohio. *Asst. Prof. of Pediatrics; Sr. Instr. in Clin. Biochem.* (6, 1958)

WEGRIA, RENE, M.D., Med. Sc.D. Presbyterian Hosp., Dept. of Med., New York City 32. *Assoc. Prof.* (1, 1941; 3, 1950)

WEICHERT, CHARLES K., Ph.D. U. of Cincinnati, Cincinnati, Ohio. *Prof. of Zool.; Dean, Coll. of Arts and Sci.* (1, 1935)

WEICHSEL, MANFRED, M.D. 47-07 41 St., Long Island City 4, N. Y. (6, 1953)

WEICHSELBAUM, THEODORE E., Ph.D. Washington U., Sch. of Med., Dept. of Surgery, St. Louis 10, Mo. *Assoc. Prof. of Exptl. Surgery.* (2, 1950)

WEIGLE, WILLIAM O., Ph.D. U. of Pittsburgh Sch. of Med., Dept. of Pathol., Pittsburgh 13, Pa. *Asst. Research Prof.* (6, 1957)

WEIKEL, JOHN H., JR., Ph.D. Mead Johnson & Co., Research Labs., Evansville 21, Ind. *Sr. Pharmacologist.* (3, 1957)

WEIL, ALFRED J., M.D. The Bronx Hosp., Dept. of Bacteriol., New York City 56. *Dir. of Dept.* (6, 1940)

WEIL, ARTHUR, M.D. 115-06 Park Lane South, Kew Gardens, N. Y. (4, 1940)

WEIL, LEOPOLD, Ph.D. USDA Eastern Regional Research Lab., Philadelphia 18, Pa. *Head, Enzyme Unit.* (2, 1942)

WEIL, MARVIN LEE, M.D. 1185 71st St., Miami Beach, Fla. *Clin. Asst. Prof. Pediatrics, U. of Miami.* (6, 1950)

WEIMAR, VIRGINIA LEE, Ph.D. Columbia U., Coll. of P. & S., New York City 32. *Research Assoc.* (1, 1957)

WEINBACH, EUGENE C., Ph.D. Natl. Insts. of Health, Lab. of Tropical Diseases, Bethesda 14, Md. *Sr. Biochemist.* (2, 1956)

WEINHOUSE, SIDNEY, Ph.D. Inst. for Cancer Research, Philadelphia 11, Pa. *Chmn., Div. of Biochem.* (2, 1948)

WEIR, EVERETT G., M.D., Ph.D. 231 S. 13th Ave., Maywood, Ill. (1, 1941)

WEISER, RUSSELL S., Ph.D. U. of Washington Sch. of Med., Dept. of Microbiol., Seattle 5. *Prof. of Immunol.* (6, 1948)

WEISIGER, JAMES R., Ph.D. Natl. Research Council, Washington 25, D. C. *Professional Assoc.* (2, 1956)

WEISS, A. KURT, Ph.D. U. of Miami Sch. of Med., Coral Gables 34, Fla. *Asst. Prof. of Physiol.* (1, 1957)

WEISS, BENJAMIN, Ph.D. New York State Psychiatric Inst., New York City 32. *Asst. Prof., Columbia U.* (2, 1957)

WEISS, CHARLES, Ph.D., M.D. Albert Einstein Med. Ctr., Northern Div., Philadelphia 41, Pa. *Head, Div. of Microbiol. and Serol.* (4, 1934; 6, 1920)

WEISS, HAROLD SAMUEL, Ph.D. Rutgers, The State U., Dept. of Poultry Sci., New Brunswick, N. J. *Assoc. Prof., Poultry Physiol.* (1, 1957)

WEISS, PAUL A., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member; Prof. and Head, Lab. of Develop. Biol.* (1, 1936)

WEISS, SAMUEL B., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Asst. Prof.* (2, 1958)

WEISS, SIDNEY, Ph.D. Colgate-Palmolive Co., Research & Develop. Dept., Jersey City 2, N. J. *Research Chemist.* (2, 1956)

WEISSBACH, ARTHUR, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Biochemist.* (2, 1958)

WEISSMAN, NORMAN, Ph.D. U. of Utah Coll. of Med., Salt Lake City. *Assoc. Prof., Patbol.; Research Assoc. Prof., Biol. Chem.* (2, 1951)

WEISSMANN, BERNARD, Ph.D. U. of Illinois Coll. of Med., Dept. of Biol. Chem., Chicago 12, Ill. *Asst. Prof.* (2, 1956)

WELCH, ARNOLD D., Ph.D., M.D. Yale U. Sch. of Med., Dept. of Pharmacol., New Haven 11, Conn. *Chmn. of Dept.* (2, 1950; 3, 1942)

WELCH, HENRY, Ph.D. Food and Drug Admin. Div. of Antibiotics, Dept. of HEW, Washington 25, D. C. *Dir.* (6, 1932)

WELD, CHARLES BEECHER, M.D. Dalhousie U., Halifax, Canada. *Prof. of Physiol.* (1, 1936)

WELD, MRS. JULIA T. Cornell U. Med. Coll., Dept. of Public Health, New York City 21. *Research in Parasitol.* (6, 1920)

WELLER, THOMAS H., M.D. Harvard Sch. of Public Health, Boston, Mass. *Richard P. Strong Prof., Tropical Public Health; Dept. Head.* (6, 1952)

WELLS, HERBERT S., M.D. VA Hosp., 54th St. and 48th Ave. S., Minneapolis, Minn. *Dir. of Research* (1, 1932)

WELLS, IBERT C., Ph.D. State U. of New York, Upstate Med. Ctr., Dept. of Biochem., Syracuse 10. *Assoc. Prof.* (2, 1952)

WELLS, JOSEPH ALBERT, M.D., Ph.D. Northwestern U. Med. Sch., Chicago 11, Ill. *Prof. of Pharmacol.* (3, 1944)

WELSH, JOHN H., Ph.D. Harvard U., Biol. Labs., Cambridge 38, Mass. *Prof. of Zool.* (1, 1945)

WELT, LOUIS GORDON, M.D. U. of North Carolina Sch. of Med., Chapel Hill. *Prof. of Med.* (1, 1956)

WENDEL, HERBERT, M.D. Smith Kline & French Labs., Philadelphia 1, Pa. *Group Leader Pharmacol.* (3, 1954)

WENDEL, WILLIAM B., Ph.D. Tulane U. Sch. of Med., Dept. of Biochem., New Orleans 18, La. *Chmn. of Dept.* (2, 1932)

WENDER, SIMON H., Ph.D. U. of Oklahoma, Dept. of Chem., Norman. *Research Prof. of Biochem.* (2, 1953)

WERBER, ERNA A., Ph.D. 170 W. 73rd St., Apt. 3D, New York City. *Chief Bacteriologist and Mycologist, Jacques Loewe Research Lab.* (6, 1948)

WERBIN, HAROLD, Ph.D. U. of California, Dept. of Physiol., Berkeley 4. *Asst. Research Physiologist (III).* (2, 1957)

WERDER, ALVAR ARVID, Ph.D. U. of Kansas Sch. of Med., Dept. of Med. Microbiol., Kansas City 12. *Prof.* (4, 1956)

WERKMAN, C. H., Ph.D. Iowa State Coll., Dept. of Bacteriol., Science Hall, Ames. *Prof.* (2, 1942)

WERLE, JACOB M., M.D. 20911 Lorain Rd., Cleveland 26, Ohio. (1, 1943)

WERNER, GERHARD, M.D. Cornell U. Med. Coll., New York City 21. *Assoc. Prof. of Pharmacol.* (3, 1958)

WERNER, HAROLD W., Ph.D. The Wm. S. Merrell Co., Cincinnati 15, Ohio. *Vice-Pres. in charge of Research.* (3, 1942)

WERTENBERGER, GRACE E., Ph.D. U. of Indiana Sch. of Med., Dept. of Physiol., Bloomington. *Assoc. Prof.* (1, 1943)

WERTHESSEN, NICHOLAS T., Ph.D. Southwest Fndn. for Res. and Educ., Dept. of Physiol. and Biochem., San Antonio, Tex. *Chmn.* (1, 1946)

WERTMAN, KENNETH, Ph.D. U. of Arizona, Dept. of Bacteriol., Tucson. *Prof.* (6, 1953)

WERTZ, ANNE W., Ph.D. U. of Massachusetts Sch. of Home Econ., Amherst. *Prof., Nutrition.* (5, 1950)

WESCOE, W. CLARKE, M.D. U. of Kansas Med. Ctr., Kansas City 12. *Dean; Prof. of Pharmacol. and Exptl. Med.* (3, 1949)

WESSON, LAURENCE G., Ph.D. Massachusetts Inst. of Technology, Cambridge. *Staff Member, Div. of Sponsored Research.* (2R, 1929; 3, 1932)

WESSON, LAURENCE G., JR., M.D. New York U.-Bellevue Med. Ctr., Post-Grad. Med. Sch., Dept. of Med. New York 16. *Assoc. Prof.* (1, 1949)

WEST, CLARK D., M.D. Children's Hosp. Res. Fndn., Cincinnati 29, Ohio. *Fellow; Assoc. Prof. of Pediatrics, U. of Cincinnati Coll. of Med.* (1, 1954)

WEST, EDWARD S., Ph.D. U. of Oregon Med. Sch., Portland. *Prof. of Biochem.* (2, 1925)

WEST, HAROLD D., Ph.D. Meharry Med. Coll., Dept. of Biochem., Nashville 8, Tenn. *Prof. and Head of Dept.* (2, 1946)

WEST, THEODORE C., Ph.D. U. of Washington, Seattle 5. *Asst. Prof. of Pharmacol.* (3, 1955)

WESTERFELD, WILFRED WIEDEY, Ph.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Prof. of Biochem.* (2, 1944)

WESTFALL, B. A., Ph.D. U. of Missouri Sch. of Med., Dept. of Physiol. and Pharmacol., Columbia. *Prof. and Chmn.* (1, 1949; 3, 1950)

WESTHEIMER, FRANK H., Ph.D. Harvard U., Cambridge 38, Mass. *Prof. of Chem.* (2, 1957)

WESTON, JEAN KENDRICK, M.D. Parke, Davis & Co., Detroit 32, Mich. *Dir., Dept. of Clin. Invest.* (4, 1956)

WESTON, RAYMOND E., M.D., Ph.D. 315 So. Elm Dr., Beverly Hills, Calif. (1, 1947)

WESTPHAL, ULRICH F., Ph.D. Army Med. Res. Lab., Fort Knox, Ky. *Chief, Protein & Steroid Sec.; Assoc. Prof. Biochem., U. of Louisville.* (2, 1952)

WETZEL, NORMAN C., M.D. 2358 Ardleigh Dr., Cleveland Heights 6, Ohio. *Consultant in Growth and Nutrition.* (5, 1952)

WEXLER, BERNARD C., Ph.D. U. of Cincinnati Coll. of Med., Cincinnati, Ohio. *Asst. Prof. of Exptl. Pathol.* (4, 1958)

WEYMOUTH, FRANK W., Ph.D. Stanford U., Calif. *Prof. of Physiol. Emer.* (1R, 1917)

WHEELER, ALBERT H., Dr.P.H. U. of Michigan Hosp., Dept. of Dermatol., Ann Arbor. *Asst. Prof.* (6, 1950)

WHEELER, GEORGE W., M.D. 235 E. 73rd St., New York City 21. *Clin. Spec., New York City Dept. of Health.* (6R, 1920)

WHEELER, MARY W., M.A. New York State Dept. of Health, Div. of Labs. and Research, Albany. *Assoc. Bacteriologist.* (6, 1933)

WHEELON, HOMER, M.D. The Polyclinic, 920-2nd Ave., Seattle 4, Wash. (1, 1919)

WHIPPLE, GEORGE H., M.D., Sc.D. U. of Rochester

Sch. of Med. and Dent., Rochester 20, N.Y. *Prof. of Pathol. Emer.* (1R, 1911; 4R, 1913)

WHISTLER, ROY L., Ph.D. Purdue U., Biochem. Dept., Lafayette, Ind. *Asst. Head of Dept.* (2, 1957)

WHITE, ABRAHAM, Ph.D. Albert Einstein Coll. of Med., Dept. of Biochem., New York City 61. *Prof. and Chmn.; Assoc. Dean.* (2, 1934; 5, 1937)

WHITE, ABRAHAM G., M.D. Mt. Sinai Hosp., New York City. *Asst. Attending Physician.* (1, 1955)

WHITE, ALAN G. C., Ph.D. Tulane U., Dept. of Biochem., New Orleans, La. *Assoc. Prof.* (2, 1949)

WHITE, CHESTER W., JR., M.D. Boston Lying-in Hosp., Boston, Mass. *Anesthesiologist-in-Chief.* (3, 1956)

WHITE, FLORENCE R., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Biochemist.* (2, 1946)

WHITE, FRANK D., Ph.D. U. of Manitoba, Faculty of Med., Winnipeg, Canada. *Prof. of Biochem.* (2R, 1931)

WHITE, HARVEY LESTER, M.D. Washington U. Sch. of Med., St. Louis 10, Mo. *Prof. of Physiol.* (1, 1923)

WHITE, JULIUS, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chief, Lab. of Physiol.* (2, 1937)

WHITE, PAUL DUDLEY, M.D. 264 Beacon St., Boston, Mass. (3, 1921)

WHITE, PHILIP L., Sc.D. American Med. Assn., 535 N. Dearborn St., Chicago 10, Ill. *Secy., Council on Foods and Nutrition.* (5, 1957)

WHITE, WILFRID F., M.S. Armour and Co., Research Div., Chicago 9, Ill. *Head, Biochem. Sec.* (2, 1957)

WHITEHAIR, C. K., D.V.M., Ph.D. Michigan State U., East Lansing. *Prof., Animal Pathol.* (5, 1952)

WHITEHEAD, RICHARD W., M.D. U. of Colorado Sch. of Med., Denver 20. *Prof. of Pharmacol.* (1, 1933; 3, 1928)

WHITEHORN, WILLIAM V., M.D. U. of Illinois Coll. of Med., Chicago. *Prof. of Physiol.* (1, 1947)

WHITELEY, H. R., Ph.D. U. of Washington, Dept. of Microbiol., Seattle. *Res. Instr.* (2, 1958)

WHITNEY, JOHN E., Ph.D. U. of Arkansas Med. Ctr., Dept. of Physiol., Little Rock. *Asst. Prof.* (1, 1958)

WHITTENBERGER, JAMES L., M.D. Harvard Sch. of Public Health, Dept. of Physiol., Boston 15, Mass. *Prof. and Head.* (1, 1949)

WHITTLESEY, PHILIP, M.D. Johns Hopkins U. Sch. of Med., Dept. of Med., Baltimore 5, Md. (3, 1955)

WICK, ARNE N., Ph.D. 615 Rosemont St., La Jolla, Calif. *San Diego State Coll.* (2, 1951)

WIEDEMAN, MARY PURCELL, Ph.D. Temple U. Sch. of Med., Philadelphia 40, Pa. *Asst. Prof. of Physiol.* (1, 1956)

WIENER, ALEXANDER S., M.D. 64 Rutland Rd., Brooklyn, N.Y. *Sr. Bacteriologist, Office Chief Med. Exam. N.Y.C.* (6, 1932)

WIERCINSKI, FLOYD J., Ph.D. Drexel Inst. of Technology, Dept. of Biol. Sci., Philadelphia 4, Pa. *Assoc. Prof.* (1, 1950)

WIERSMA, CORNELIS A. G., Ph.D. California Inst. of Technology, Biology Div., Pasadena. *Prof. of Physiol.* (1, 1941)

WIESE, ALVIN C., Ph.D. U. of Idaho, Dept. of Agric. Chem., Moscow. *Prof. and Head of Dept.* (5, 1950)

WIESE, HILDA F., Ph.D. U. of Texas Med. Sch., Galveston. *Prof. and Consultant in Pediatric Nutrition.* (5, 1952)

WIEST, WALTER G., Ph.D. U. of Utah Coll. of Med., Dept. of Biol. Chem., Salt Lake City 12. *Asst. Prof.* (2, 1958)

WIGGERS, CARL J., M.D., Sc.D. 1360 Cleveland Hts. Blvd., Cleveland 21, Ohio. *Prof. Emer., Physiol., Western Reserve U.* (1R, 1907; 3R, 1909)

WIGGERS, HAROLD C., Ph.D. Albany Med. Coll., Albany 8, N.Y. *Dean; Prof. of Physiol.* (1, 1938)

WIGODSKY, HERMAN S., Ph.D., M.D. 420 E. Houston St., San Antonio 5, Tex. *Dir., San Antonio Div., U. of Texas Post-Grad. Med. Sch.* (1, 1943)

WIKLER, ABRAHAM, M.D. NIMH Addiction Research Ctr., PHS Hosp., Lexington, Kr. *Chief, Neuropsychiatric Sec.* (3, 1944)

WILBER, CHARLES G., Ph.D. Chem. Warfare Labs., Comparative Physiol. Br., Army Chemical Center, Md. *Chief.* (1, 1947)

WILBRANDT, WALTHER, M.D. U. of Berne, Dept. of Pharmacol., Berne, Switzerland. *Prof.* (3, 1956)

WILBUR, KARL M., Ph.D. Duke U., Dept. of Zool., Durham, N.C. *Prof.* (1, 1951)

WILCOX, PHILIP E., Ph.D. U. of Washington Sch. of Med., Dept. of Biochem., Seattle 5. *Assoc. Prof.* (2, 1957)

WILDE, WALTER S., Ph.D. U. of Michigan Sch. of Med., Ann Arbor. *Prof. of Physiol.* (1, 1944)

WILDER, RUSSELL M., Ph.D., M.D. Mayo Clinic, 705 Eighth Ave., S.W., Rochester, Minn. (1R, 1921; 4R, 1924; 5R, 1933)

WILENS, SIGMUND L. New York U.-Bellevue Med. Ctr., New York City 16. *Prof. and Dir. of Pathol.* (4, 1952)

WILEY, FRANK H., Ph.D. Food and Drug Admin., Dept. of HEW, Washington 25, D.C. *Chief, Div. of Pharmaceutical Chem.* (2, 1933)

WILHELMI, ALFRED E., Ph.D. Emory U. School of Med., Dept. of Biochem., Atlanta 22, Ga. *Prof. and Chmn. of Dept.* (2, 1942)

WILHELMJ, CHARLES MARTEL, M.D. Creighton U. Sch. of Med., Omaha, Nebr. *Prof. of Physiol.* (1, 1931)

WILKERSON, VERNON A., M.D., Ph.D. 61 K St., N.W., Washington, D.C. *Visiting Prof., Dept. of Biochem., Howard U. Sch. Med.* (2, 1936)

WILLIAMS, CARROLL M., Ph.D., M.D. Harvard U., Biol. Labs., Cambridge, Mass. *Prof. of Zool.* (1, 1947)

WILLIAMS, GEORGE Z., M.D., Natl. Insts. of Health, Clin. Ctr., Dept. of Clin. Pathol., Bethesda 14, Md. *Chief.* (4, 1952)

WILLIAMS, HAROLD H., Ph.D. Cornell U., Dept. of Biochem. and Nutrition, Savage Hall, Ithaca, N.Y. *Prof. and Head.* (2, 1938; 5, 1936)

WILLIAMS, J. W., Ph.D. U. of Wisconsin, Chem. Bldg., Madison. *Prof. of Chem.* (2, 1944)

WILLIAMS, JESSE N., JR., Ph.D. U. of Wisconsin, Dept. of Biochem., Madison 6. *Assoc. Prof.* (2, 1951)

WILLIAMS, M. HENRY, JR., M.D. Grasslands Hosp. Valhalla, N.Y. *Dir., Cardio-Resp. Lab.* (1, 1955)

WILLIAMS, MAUDE F., Ph.D. U. of North Carolina

Woman's Coll., Dept. of Biol., Greensboro. *Assoc. Prof. of Physiol.* (1, 1955)

WILLIAMS, RAY D., M.D. 114 No. Taylor Ave., St. Louis 8, Mo. *Asst. Prof. of Clin. Med., Washington U.* (5, 1941)

WILLIAMS, RICHARD B., JR., M.D. Naval Med. Research Inst., Pathol. Div., Bethesda 14, Md. *Head.* '4, 1955)

WILLIAMS, ROBERT HARDIN, M.D. U. of Washington, Dept. of Med., Seattle. *Prof. and Exec. Officer.* (4, 1940)

WILLIAMS, ROBERT R., D.Sc. 297 Summit Ave., Summit, N. J. (2R, 1919; 5, 1941)

WILLIAMS, ROGER J., Ph.D. U. of Texas, Dept. of Chem., Austin. *Prof.; Dir., Clayton Fndn., Biochem. Inst.* (2, 1931)

WILLIAMS, VIRGINIA R., Ph.D. Louisiana State U., Food Preservation Bldg., Baton Rouge. *Assoc. Prof.* (2, 1954)

WILLIAMS, W. LANE, Ph.D. U. of Mississippi Med. Ctr., Dept. of Anatomy, Jackson. *Prof.* (1, 1947; 4, 1947)

WILLIAMS, WILLIAM L., Ph.D. American Cyanamid Co. Research Div., Pearl River, N. Y. (2, 1951)

WILLIAMS, WILLIAM L., M.D. U. of Tennessee, Dept. of Pathol. and Microbiol., Memphis. *Asst. Prof.* (4, 1956)

WILLIAMS–ASHMAN, H. G., Ph.D. U. of Chicago, Ben May Lab. for Cancer Research, Chicago 37, Ill. *Assoc. Prof. of Biochem.* (2, 1957)

WILLIAMSON, MARTIN B., Ph.D. Loyola U., Stritch Sch. of Med., Dept. of Biochem., Chicago 12, Ill. *Prof.* (2, 1953)

WILLMON, THOMAS L., M.D. Natl. Multiple Sclerosis Soc., 257 Fourth Ave., New York City 10. *Med. and Research Dir.* (1, 1951)

WILLS, J. H., Ph.D. Chem. Warfare Labs., Directorate of Med. Res., Army Chemical Center, Md. *Asst. Chief, Physiol. Div.* (1, 1943; 3, 1951)

WILSON, ARMINE T. Alfred I. duPont Inst., Wilmington 99, Del. *Chief Bacteriologist.* (6, 1949)

WILSON, D. WRIGHT, Ph.D. U. of Pennsylvania Sch. of Med. Philadelphia 4. *Rush Prof. Physiol. Chem. Emer.* (1R, 1915; 2, 1915)

WILSON, EVA D., Ph.D. 256 E. Irvin Ave., State College, Pa. *Prof., Dept. Foods and Nutr., Coll. of Home Econ., Pennsylvania State U.* (5, 1947)

WILSON, HILDEGARD N., Ph.D. Natl. Inst. of Arthritis and Metabolic Diseases, NIH, Bethesda 14, Md. *Analytical Biochemist.* (2, 1955)

WILSON, IRWIN B., Ph.D. 34 Garwood Rd., Fair Lawn, N. J. *Assoc. Prof., Columbia U., Coll. of P. & S.* (2, 1952)

WILSON, J. WALTER, Ph.D. Brown U., Dept. of Biol., Providence 12, R. I. *Chmn. of Dept.; F. L. Day Prof.* (1, 1949; 4, 1950)

WILSON, JOHN W., Ph.D. Aero Med. Res. Lab., Wright-Patterson AFB, Dayton, Ohio. *Research Physiologist.* (1, 1948)

WILSON, LOUISE PALMER, Ph.D. Wellesley Coll., Wellesley, Mass. *Prof. of Physiol.* (1, 1956)

WILSON, P. W., Ph.D. U. of Wisconsin, Dept. of Bacteriol., Madison 6. *Prof.* (2, 1939)

WILSON, ROBERT H., Ph.D. USDA, Western Utilization Res. and Develop. Div., Albany 10, Calif. *Prin. Pharmacologist.* (3, 1937)

WILSON, T. HASTINGS, Ph.D., M.D. Harvard Med. Sch., Dept. of Physiol., Boston 15, Mass. *Assoc.* (2, 1957)

WILSON, VICTOR J., Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Research Assoc.* (1, 1957)

WILSON, WALTER L., Ph.D. U. of Vermont Coll. of Med., Burlington. *Asst. Prof. of Physiol. and Biophysics.* (1, 1954)

WINBURY, MARTIN M., Ph.D. Schering Corp., Pharmacol. Dept., Bloomfield, N. J. *Sr. Pharmacologist.* (3, 1952)

WINDER, CLAUDE V., Sc.D. Parke, Davis & Co., Detroit 32, Mich. *Research Pharmacologist.* (1, 1938; 3, 1948)

WINDLE, WILLIAM FREDERICK, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Chief, Lab. of Neuroanatomical Sci.* (1, 1937)

WINGO, WILLIAM J., Ph.D. 1924 Saulter Rd., Birmingham 9, Ala. *Assoc. Prof., U. of Alabama. Med. Coll.* (2, 1953)

WINITZ, MILTON, Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Chemist.* (2, 1956)

WINNICK, THEODORE, Ph.D. Weizmann Inst. of Sci., Sec. of Biochem., Rehovoth, Israel. *Prof. and Head of Dept.* (2, 1946)

WINTER, CHARLES A., Ph.D. Merck Inst. for Therap. Research, West Point, Pa. *Dir. of Physiol.* (1, 1940; 3, 1952)

WINTER, IRWIN CLINTON, Ph.D., M.D. G. D. Searle & Co., P. O. Box 5110, Chicago 80, Ill. *Dir. of Clin. Research.* (3, 1941)

WINTERNITZ, M. C., M.D. 2126 Connecticut Ave., Washington 8, D. C. (4R, 1913)

WINTERS, WALLACE D., M.D., Ph.D. Marquette U. Sch. of Med. Dept. of Pharmacol., Milwaukee 3, Wis. *Instr.* (3, 1956)

WINTERSTEINER, OSKAR, Ph.D. Squibb Inst. for Med. Research, Div. of Organic Chem., New Brunswick, N. J. *Dir.* (2, 1930)

WINTROBE, M. M., M.D., Ph.D. 175 E. 21st St., S., Salt Lake City. *Prof. & Head, Dept. Int. Med., U. Utah: Dir., Lab. Hered. & Metabol. Disorders.* (4, 1940)

WINZLER, RICHARD J., Ph.D. U. of Illinois Coll. of Med., Dept. of Biochem., Chicago 12. *Prof. and Head.* (2, 1946)

WISE, GEORGE H., Ph.D. North Carolina State Coll., Dept. of Animal Industry, Raleigh. *Prof. and Head, Animal Nutrition Sec.* (5, 1951)

WISEMAN, BRUCE KENNETH, M.D. Ohio State U., Dept. of Med., Kinsman Hall, Columbus. *Prof. and Chmn. of Dept.* (4, 1932)

WISSEMAN, CHARLES L., JR., M.D. U. of Maryland Sch. of Med., Dept. of Microbiol., Baltimore 1. *Prof. and Head of Dept.* (6, 1952)

WISSLER, ROBERT W., M.D., Ph.D. U. of Chicago, Chicago 37, Ill. *Prof. of Pathol.* (4, 1949; 6, 1955)

WITEBSKY, ERNEST, M.D. U. of Buffalo Sch. of Med., Dept. of Bacteriol. and Immunol., Buffalo 14, N. Y. *Dist. Prof. and Head of Dept.* (6, 1935)

WITT, PETER N., M.D. State U. of New York, Upstate Med. Ctr., Syracuse 10. *Asst. Prof. of Pharmacol.* (3, 1957)

WITTENBERG, JONATHAN B., Ph.D. Albert Einstein Coll. of Med., Dept. of Physiol., New York

City 61. *Asst. Prof. of Physiol.* (2, 1957)

WITTER, ROBERT F., Ph.D. PHS Communicable Disease Ctr., Toxicol. Sec., P. O. Box 769, Savannah, Ga. *Chief Biochemist.* (2, 1951)

WITTICH, FRED W., M.D. 3230 Alabama Ave., Minneapolis 16, Minn. *Ed., Rev. of Allergy and Applied Immunol.* (6, 1944)

WIXOM, ROBERT L., Ph.D. U. of Arkansas Sch. of Med., Dept. of Biochem., Little Rock. *Asst. Prof.* (5, 1958)

WOHL, MICHAEL G., M.D. Hahnemann Med. Coll., Div. of Biol. Chem., Philadelphia 3, Pa. *Chief of Human Nutrition.* (5, 1953)

WOLF, ABNER, M.D. Columbia U., Coll. of P. & S., New York City 32. *Prof. of Neuropathol.* (4, 1948)

WOLF, ARNOLD VERYL, Ph.D. Walter Reed Army Inst. of Research, Dept. Cardioresp. Diseases, Washington 12, D.C. *Chief, Renal Sec.* (1, 1946)

WOLF, GEORGE, D.Phil. U. of Illinois, 157 Davenport Hall, Urbana. *Assoc. Prof. of Animal Nutrition.* (2, 1957)

WOLF, STEWART, M.D. 644 N. E. 14th St., Oklahoma City, Okla. *Prof. and Head, Dept. of Med., Oklahoma U.* (1, 1948)

WOLFE, HAROLD RECLUS, Ph.D. U. of Wisconsin, Madison 6. *Prof. of Zool.* (6, 1952)

WOLFE, JACK B., Ph.D. U. of Southern California Sch. of Med., Dept. of Med. Microbiol., Los Angeles 7. *Asst. Prof.* (2, 1958)

WOLFF, HAROLD G., M.D. New York Hosp., New York City 21. *Attend. Physician; Prof. of Med. (Neurol.), Cornell U.* (1, 1930; 3, 1942)

WOLFF, WILLIAM A., Ph.D. Bowman Gray Sch. of Med., Winston-Salem 7, N.C. *Assoc. Prof. of Clin. Chem. and Toxicol.* (2, 1947)

WOLFROM, M.L., Ph.D. Ohio State U., Dept. of Chem., Columbus 10. *Prof.* (2, 1953)

WOLL, EPHRAIM, M.D. The Danbury Hosp., Danbury, Conn. (4, 1951)

WOLLENBERGER, ALBERT, Ph.D. German Acad. of Sci., Lab. of Circulation Research, Berlin-Buch, Germany. (3, 1951)

WOLLMAN, SEYMOUR H., Ph.D. Natl. Cancer Inst., Lab. of Physiol., NIH, Bethesda 14, Md. *Biophysicist.* (1, 1955)

WOLTERINK, LESTER F., Ph.D. Michigan State U., Dept. of Physiol., East Lansing. *Prof.* (1, 1951)

WOMACK, C. RAY, M.D. Louisiana State U., Sch. of Med., New Orleans 12. *Assoc. Prof. of Microbiol.; Asst. Prof. of Med.* (4, 1955)

WOMACK, MADELYN, Ph.D. USDA, Human Nutrition Research Br., Inst. of Home Econ., Washington 25, D.C. *Biochemist.* (5, 1947)

WONG, HARRY Y. C., Ph.D. 7211 Hilton Ave., Takoma Park 12, Md. *Asst. Prof. of Physiol., Howard U. Sch. of Med.* (1, 1956)

WOOD, DEREK RAWLINS, B.M., B.Ch. McGill U., Dept. of Pharmacol., Montreal 2, Canada. *Assoc. Prof.* (3, 1958)

WOOD, EARL H., Ph.D., M.D. Mayo Fndn., Rochester, Minn. *Prof., Physiol., Grad Sch., U. Minn.; Consultant Physiol., Mayo Clinic.* (1, 1943; 3, 1949)

WOOD, HARLAND G., Ph.D. Western Reserve U., Dept. of Biochem., Cleveland 6, Ohio. *Prof. and Chmn. of Dept.* (2, 1944)

WOOD, HARRISON F., M.D. Irvington House, Irvington-on-Hudson, N.Y. *Med. Dir.* (6, 1954)

WOOD, JOHN L., Ph.D. U. of Tennessee Sch. of Biol. Sci., Div. of Chem., Memphis 3. *Prof. and Chief.* (2, 1947)

WOOD, SUMNER, JR., M.D. USAF Sch. of Aviation Med., Randolph AFB, Tex. *Pathologist.* (4, 1958)

WOOD, THOMAS R., Ph.D. E. I. duPont de Nemours & Co., Inc., Newark, Del. *Mgr., Stine Lab.* (2, 1957)

WOOD, WILLIS AVERY, Ph.D. U. of Illinois, 460 Animal Sci. Lab., Urbana. *Assoc. Prof., Dairy Bacteriol.* (2, 1954)

WOODARD, GEOFFREY, Ph.D. RFD #3, Herndon, Va. *Pharmacologist, Woodard Research.* (3, 1953)

WOODBURY, DIXON M., Ph.D. U. of Utah Coll. of Med., Dept. of Pharmacol., Salt Lake City 12. *Assoc. Research Prof.* (3, 1951)

WOODBURY, J. WALTER, Ph.D. U. of Washington, Sch. of Med., Dept. of Physiol. and Biophysics, Seattle 5. *Assoc. Prof.* (1, 1952)

WOODBURY, LOWELL A., Ph.D. 248 University St., Salt Lake City 2, Utah. (1, 1951)

WOODBURY, ROBERT A., Ph.D., M.D. U. of Tennessee, Div. of Pharmacol., Memphis. *Prof. and Chief.* (1, 1936; 3, 1941)

WOODCOCK, ALAN H., Ph.D. QM Res. and Engin. Command, Natick, Mass. *Head, Biophysics Br.* (1, 1952)

WOODRUFF, CALVIN W., M.D. Vanderbilt U. Sch. of Med., Nashville 5, Tenn. *Assoc. Prof. of Pediatrics.* (5, 1954)

WOODS, ALAN C., M.D., Johns Hopkins Hosp., Wilmer Ophthalmol. Inst., Baltimore, Md. *Emer. Prof. of Ophthalmol., Johns Hopkins U.* (6, 1918)

WOODS, ELLA, Ph.D. 1416 N. 13th St., Boise, Idaho. *Home Economist Emer., U. of Idaho.* (2R, 1925; 5R, 1933)

WOODS, EUGENE F., Ph.D. Med. Coll. of South Carolina, Charleston 16. *Assoc. in Pharmacol.* (3, 1958)

WOODS, LAUREN A., M.D., Ph.D. U. of Michigan, Dept. of Pharmacol., Ann Arbor. *Prof.* (3, 1949)

WOODS, MARK W., Ph.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. *Cytologist.* (2, 1954)

WOODWARD, ALVALYN E., Ph.D. Bay View Acres, Mt. Pleasant, S.C. *Asst. Prof. Emer. of Zool., U. of Michigan.* (1R, 1932)

WOODWARD, EDWARD ROY, M.D. U. of Florida Coll. of Med., Dept. of Surgery, Gainesville. *Prof. and Chmn. of Dept.* (1, 1957)

WOOLLEY, D. WAYNE, Ph.D. Rockefeller Inst. for Med. Research, New York City 21. *Member and Prof.* (2, 1946; 3, 1957; 5, 1941)

WOOLSEY, CLINTON N., M.D. U. of Wisconsin, 214 S. M. I., Madison 6. *Charles Sumner Slichter Prof. of Neurophysiol.* (1, 1938)

WORKMAN, WILLIAM G., M.D. Natl. Insts. of Health, Bethesda 14, Md. *Med. Dir., PHS.* (6, 1952)

WORTIS, S. BERNARD, M.D. New York U. Coll. Med., Psychiat. & Neurol., New York 16. *Prof. & Chmn., Dir. Psychiat. & Neurol. Bellevue Med. Ctr.* (1, 1947)

WOSILAIT, WALTER D., Ph.D. State U. of New York Coll. of Med., Brooklyn 3. *Asst. Prof. of Pharmacol.* (2, 1957)

WOTIZ, HERBERT H., Ph.D. Boston U. Sch. of Med., Boston, Mass. *Assoc. Research Prof.* (2, 1956)

WRIGHT, ANGUS, M.D. 1414 S. Hope St., Los Angeles 15, Calif. *Pathologist, California Hosp.* (4, 1935)

WRIGHT, ARTHUR W., M.D. Albany Med. Coll., Albany 8, N.Y. *Prof. of Patbol.* (4, 1941)

WRIGHT, BARBARA E., Ph.D. Natl. Heart Inst., NIH, Bethesda 14, Md. *Microbiologist.* (2, 1958)

WRIGHT, CHARLES INGHAM, Ph.D. Natl. Insts. of Health, Bethesda 14, Md. *Sr. Pharmacologist, PHS.* (1, 1935; 3, 1936)

WRIGHT, CLAUDE-STARR, M.D. Med. Coll. of Georgia, Dept. of Med., Augusta. *Prof.* (6, 1953)

WRIGHT, ERNEST B., Ph.D. U. of Florida Coll. of Med., Gainesville. *Assoc. Prof. of Physiol.* (1, 1952)

WRIGHT, GEORGE G., Ph.D. Fort Detrick, Frederick, Md. *Chief, Immunol. Br., Med. Invest. Div.* (6, 1943)

WRIGHT, GEORGE W., M.D. St. Luke's Hosp., Dept. of Med. Research, 11311 Shaker Blvd., Cleveland 4, Ohio. *Head of Dept.* (1, 1952)

WRIGHT, HAROLD N., Ph.D. U. of Minnesota, Minneapolis 14. *Prof. of Pharmacol.* (3, 1931)

WRIGHT, LEMUEL D., Ph.D. Cornell U., Savage Hall, Ithaca, N.Y. *Prof. of Nutrition.* (2, 1946; 5, 1946)

WRIGHT, PAUL A., Ph.D. U. of New Hampshire, Dept. of Zool., Durham. *Assoc. Prof.* (1, 1950)

WRIGHT, SYDNEY L., Ph.D. Fox Hill Farm, Jamestown, R.I. (2, 1933)

WU, HSEIN, Ph.D. 28 Adams St., Brookline, Mass. (2, 1950)

WULFF, V. J., Ph.D. Syracuse U., Dept. of Zool., Syracuse, N.Y. *Prof. and Chmn. of Dept.* (1, 1949)

WULZEN, ROSALIND, Ph.D. Oregon State Coll., Corvallis. *Prof. Emer. of Zool.* (1R, 1916)

WUNDER, CHARLES C., Ph.D. State U. of Iowa Coll. of Med., Dept. of Physiol., Iowa City. *Asst. Prof.* (1, 1957)

WYATT, GERARD R., Ph.D. Yale U. Sch. of Med., Dept. of Biochem., New Haven 11, Conn. *Asst. Prof.* (2, 1956)

WYMAN, LELAND C., Ph.D. Boston U., Coll. of Liberal Arts, 675 Commonwealth Ave., Boston, Mass. *Prof. of Biol.* (1, 1927)

WYNN, WINFREY, M.S., M.A. Emory U., Div. of Basic Health Sci., Atlanta 22, Ga. *Assoc. Prof. of Physiol.* (1, 1957)

WYNNE, ARTHUR M., Ph.D. U. of Toronto, Dept. of Biochem., Toronto 5, Canada. *Prof. and Head of Dept.* (2, 1940)

WYSS, ORVILLE, Ph.D. U. of Texas, Dept. of Bacteriol., Austin. *Prof.* (2, 1951)

YAGER, ROBERT H., V.M.D. USA Med. Unit, Fort Detrick, Md. *Asst. to Commanding Officer.* (6, 1950)

YAGI, YASUO, D.Sc. Roswell Park Mem. Inst., Buffalo 3, N.Y. *Sr. Cancer Research Scientist.* (6, 1957)

YANOFSKY, CHARLES, Ph.D. Stanford U., Dept. of Biol. Sci., Stanford, Calif. *Assoc. Prof.* (2, 1958)

YATES, FRANCIS EUGENE, M.D. Harvard Med. Sch., Dept. of Physiol., Boston 15, Mass. *Assoc. in Physiol.* (1, 1957)

YONKMAN, FREDRICK F., Ph.D., M.D., D.Sc. Ciba Pharmaceutical Products, Inc., Summit, N.J. *Vice-Pres. in charge of Research.* (3, 1931)

YOUMANS, GUY P., M.D., Ph.D. Northwestern U. Med. Sch., Dept. of Microbiol., Chicago, Ill. *Prof. and Head of Dept.* (6, 1955)

YOUMANS, JOHN B., M.D. Dept. of the Army, Office of Surgeon General, Washington 25, D.C. *Tech. Dir. of Research.* (5, 1948)

YOUMANS, WILLIAM BARTON, Ph.D., M.D. U. of Wisconsin Med. Sch., Dept. of Physiol., Madison 6. *Prof. and Chmn. of Dept.* (1, 1939; 3, 1949)

YOUNG, A. G., Ph.D., M.D. 520 Commonwealth Ave., Boston 15, Mass. (3, 1925)

YOUNG, ALLAN C., Ph.D. U. of Washington, Dept. of Physiol. and Biophysics, Seattle 5. *Assoc. Prof.* (1, 1952)

YOUNG, CHARLOTTE M., Ph.D. Cornell U. Grad. Sch. of Nutrition, Ithaca, N.Y. *Prof. of Med. Nutrition.* (5, 1956)

YOUNG, E. G., Ph.D. Natl. Research Council of Canada, Atlantic Regional Lab., Halifax, Canada. *Dir.* (2, 1925)

YOUNG, JOSEPH M., M.D. Kennedy VA Hosp., Memphis 15, Tenn. *Chief, Lab. Serv.* (4, 1955)

YOUNG, ROBERT J., Ph.D. The Buckeye Cellulose Corp., MA&R Bldg., Cincinnati 17, Ohio. *Res. Biochemist.* (5, 1958)

YOUNGNER, JULIUS STUART, Sc.D. U. of Pittsburgh Sch. of Med., Dept. of Microbiol., Pittsburgh, Pa. *Assoc. Prof.* (6, 1950)

YU, TSAI-FAN, M.D. Mt. Sinai Hosp., New York City 29. *Res. Assoc. in Med.; Asst. Prof. in Med., Columbia U.* (1, 1958; 3, 1958)

YUILE, CHARLES L., M.D., C.M. U. of California Sch. of Med., Los Angeles 24. *Prof. of Patbol.* (4, 1941)

ZABIN, IRVING, Ph.D. U. of California Sch. of Med., Dept. of Physiol. Chem., Los Angeles 24. *Asst. Prof.* (2, 1953)

ZAFFARONI, ALEJANDRO, Ph.D. P.O. Box 2679, Mexico, D.F. *Exec. Vice-Pres., Syntex, S. A.* (2, 1954)

ZAMCHECK, NORMAN, M.D. Boston City Hosp., Mallory Inst. of Pathol., Boston, Mass. *Res. Physician, G. I. Research Lab.* (4, 1954)

ZAMECNIK, PAUL, M.D. Massachusetts Gen. Hosp., Boston 14, Mass. *Prof. of Med., Harvard U.* (2, 1950)

ZAMENHOF, STEPHEN, Ph.D. Columbia U., Coll. of P. & S., Dept. of Biochem., New York City 32. *Assoc. Prof.* (2, 1951)

ZAPATA-ORTIZ, VICENTE, M.D., D.M. Univ. de San Marcos, Fac. of Med., Apartado 2694, Lima, Peru. *Prof. of Pharmacol.* (3, 1951)

ZAPP, JOHN A., JR., Ph.D. Haskell Lab., E. I. duPont de Nemours & Co., Newark, Del. *Dir.* (1, 1950)

ZARROW, M. X., Ph.D. Purdue U., Dept. of Biol. Sci., Lafayette, Ind. *Prof. of Zool.* (1, 1949)

ZAUDER, HOWARD L., M.D., Ph.D. Albert Einstein Coll. of Med., New York City 61. *Asst. Prof. of Anesthesiol.* (3, 1955)

ZECHMEISTER, L., Dr. Ing. California Inst. of Tech., Pasadena. *Prof. of Organic Chem., (Retired).*

(2, 1941)

ZECKWER, ISOLDE T., M.D. U. of Pennsylvania Sch. of Med.. Philadelphia 4. *Prof. of Patbol.* (1R, 1934; 4R, 1927)

ZEIDMAN, IRVING, M.D. U. of Pennsylvania Sch. of Med., Dept. of Pathol., Philadelphia 4. *Assoc. Prof.* (4, 1946)

ZELDIS, LOUIS J., M.D. U. of California Med. Ctr., Dept. of Pathol., Los Angeles 24. *Prof.* (4, 1945)

ZELITCH, ISRAEL, Ph.D. Connecticut Agric. Expt. Sta., P. O. Box 1106, New Haven 4. *Assoc. Biochemist.* (2, 1954)

ZELLER, E. ALBERT, M.D., Ph.D. Northwestern U. Med. Sch., Chicago 11. *Prof. of Biochem.* (2, 1951)

ZICHIS, JOSEPH, Ph.D. Markham Labs., 9246 S. Vincennes Ave., Chicago 20, Ill. *Dir.* (6, 1951)

ZIERLER, KENNETH L., M.D. Johns Hopkins Hosp., Baltimore 5, Md. *Assoc. Prof. in Med., Johns Hopkins U.* (1, 1951)

ZILL, LEONARD P., Ph.D. Research Inst. for Advanced Studies, Inc., Baltimore 12, Md. *Sr. Scientist.* (2, 1957)

ZILLIKEN, FRIEDRICH, Ph.D. U. of Pennsylvania Sch. of Med., Philadelphia 4. *Asst. Prof. of Biochem.* (2, 1957)

ZILVERSMIT, D. B., Ph.D. U. of Tennessee, Dept. of Physiol., Memphis 3. *Prof.* (1, 1949)

ZIMMERMAN, BERNARD, M.D., Ph.D. U. of Minnesota Med. Sch., Minneapolis 14. *Assoc. Prof. of Surgery.* (4, 1958)

ZIMMERMAN, HARRY M., M.D. Montefiore Hosp., New York City 67. *Chief, Lab. Div.; Prof. of Pathol., Columbia U.* (4, 1933)

ZIPKIN, ISADORE, Ph.D. Natl. Inst. of Dental Res., Lab. of Oral and Biol. Chem.. NIH, Bethesda 14, Md. *Asst. Chief.* (5, 1958)

ZIRKLE, RAYMOND E., Ph.D. U. of Chicago, Committee on Biophysics, Chicago, Ill. *Prof. of Radiobiol.* (1, 1948)

ZITTLE, CHARLES A., Ph.D. USDA, Eastern Regional Research Lab., Philadelphia 18, Pa. *Sec. Chief, Protein Div.* (2, 1946)

ZUBROD, CHARLES G., M.D. Natl. Cancer Inst., NIH, Bethesda 14, Md. (3, 1952)

ZUCKER, MARJORIE B., Ph.D. Sloan-Kettering Inst. for Cancer Research, New York City 21. *Assoc.* (1, 1947)

ZWEIFACH, BENJAMIN W., Ph.D. New York U. Coll. of Med., Dept. of Pathol., New York City 16. (1, 1945)

ZWEMER, RAYMUND L., Ph.D. UNESCO, Div. of Internatl. Cooperation for Scientific Research, Paris 16,[e] France. *Chief.* (1, 1930)

SUMMARY OF MEMBERSHIP

The American Physiological Society	1,701
American Society of Biological Chemists	1,497
American Society for Pharmacology and Experimental Therapeutics	799
The American Society for Experimental Pathology	568
American Institute of Nutrition	506
The American Association of Immunologists	540
Total Membership	5,611

DECEASED MEMBERS

ALGIRE, GLENN H. (4, 1945), April 28, 1958
BEAZELL, JAMES M. (1, 1939), February 10, 1958
BRUGER, MAURICE (2, 1935; 5, 1935), November 26, 1957
COLLINS, DEAN A. (1, 1938), December 3, 1957
CRIDER, JOSEPH O. (1R, 1935), November 5, 1957
ENGLE, EARL THERON (1, 1930), December 17, 1957
FLOSDORF, EARL W. (6, 1941), April 29, 1958
GREEN, ARDA A. (2, 1932), January, 1958
HAUGE, SIGFRED M. (2, 1954; 5, 1933), November 26, 1957
HILLER, ALMA (2R, 1929), June 19, 1958
HUDACK, STEPHEN SYLVESTER (4, 1953), May 13, 1958
KILLIAN, JOHN ALLEN (2, 1921), December 2, 1957
KOBRAK, HEINRICH G. (1, 1948), October 9, 1957
LAPICQUE, L. (1H, 1946), Died in 1952
LEVISON, LOUIS A. (6, 1916), 1958
RAKE, GEOFFREY W. (6, 1939), April 20, 1958
SAPHRA, IVAN (6, 1946), October 7, 1957
STEWART, WINIFRED B. (1, 1941), August 20, 1957
TAYLOR, CRAIG L. (1, 1945), April 24, 1958
TURNER, ABBY H. (1R, 1928), November 26, 1957
WEISS, EMIL (6R, 1927), Died in 1955
WOOD, HORATIO C., JR. (3R, 1908), March 31, 1958

GEOGRAPHICAL LIST OF MEMBERS

ALABAMA

AUBURN
W. D. Salmon, 2, 5
H. E. Sauberlich, 2, 5

BIRMINGHAM
S. B. Barker, 1, 3
J. M. Bruhn, 1
E. B. Carmichael, 1, 2
A. E. Casey, 4
L. C. Clark, Jr., 1
J. D. Emerson, 1
E. E. Evans, 6
K. Hare, 1
S. P. Kent, 4
C. D. Kochakian, 1, 2
L. L. Langley, 1
I. F. A. McManus, 4
W. W. Pigman, 2
T. W. Robinson, 1
F. M. Schabel, Jr., 4
L. H. Schneyer, 1
H. E. Skipper, 2
T. D. Spies, 3, 4, 5
R. S. Teague, 3
W. J. Wingo, 2

FAIRHOPE
R. A. Lambert, 4

MONTGOMERY
C. C. Shepard, 6

TUSCALOOSA
C. Brooks, 1, 3

UNIVERSITY
B. L. Davey, 5
F. L. Meyer, 5
E. N. Todhunter, 5

ALASKA

R. J. Hock, 1
L. Irving, 1
E. M. Scott, 2

ARIZONA

GLENDALE
B. W. Heywang, 5

PHOENIX
M. J. Blish, 2R
C. L. Hess, 1R
F. D. Mann, 1, 4

TUCSON
L. E. Booher, 2, 5
K. K. Jones, 1R
A. R. Kemmerer, 5
K. Werrman, 6

ARKANSAS

FAYETTEVILLE
M. C. Kik, 5
A. Nettleship, 4
J. Sacks, 1, 3
E. L. Stephenson, 5
B. Sure, 2R, 5R

LITTLE ROCK
S. A. Corson, 1
P. L. Day, 2, 5
J. S. Dinning, 5
C. E. Duffy, 6
T. D. Norman, 4
T. C. Panos, 4
H. G. Schlumberger, 4
L. D. Seager, 3
J. E. Whitney, 1
R. L. Wixom, 5

MENA
H. D. Lightbody, 2

CALIFORNIA

ALBANY
G. Alderton, 2
A. N. Booth, 2
E. F. Jansen, 2
J. C. Lewis, 2
H. Lineweaver, 2
F. A. Loewus, 2
H. P. Lundgren, 2
S. Schwimmer, 2
R. H. Wilson, 3

ALHAMBRA
W. M. Cahill, 2

BERKELEY
S. Abraham, 2
D. I. Arnon, 2
C. E. Ballou, 2
A. K. Balls, 2
H. A. Barker, 2
E. L. Bennett, 2
L. J. Bogert, 2R
H. C. Bradley, 1R, 2R
M. M. Brooks, 1
M. Calvin, 2
F. H. Carpenter, 2
I. L. Chaikoff, 1
S. F. Cook, 1
C. A. Dekker, 2
C. C. Delwiche, 2
E. L. Dobson, 1
M. Doudoroff, 2
S. S. Elberg, 6
H. M. Evans, 1R
D. S. Feingold, 2
H. O. L. Fischer, 2R
J. Fong, 6
H. L. Fraenkel-Conrat, 2
J. B. Gilbert, 2
F. R. Goetzl, 1
E. L. Hansen, 3
W. Z. Hassid, 2
M. R. Heinrich, 2
J. Hidalgo, 3
M. Ikawa, 2
H. B. Jones, 1
M. A. Joslyn, 2
L. S. Kelly, 1
P. L. Kirk, 2
C. A. Knight, 2
A. P. Krueger, 4, 6
E. H. Lennette, 4, 6
S. Lepkovsky, 2, 5
C. H. Li, 2
G. Mackinney, 2
M. E. McClain, 6
A. D. McLaren, 2
A. F. Morgan, 2R, 5
J. B. Neilands, 2
M. M. Nelson, 5
J. H. Northrop, 2
R. Okey, 2, 5
H. S. Olcott, 2
N. Pace, 1
A. B. Pardee, 2
E. W. Putman, 2
J. C. Rabinowitz, 2
G. Rosenfeld, 1
D. R. Sanadi, 2
H. K. Schachman, 2
V. P. Seeberg, 3
M. E. Simpson, 1
E. E. Snell, 2, 5
R. Y. Stanier, 2
W. M. Stanley, 2, 6
E. S. Sundstroem, 2R
A. Taurog, 2
P. S. Timiras, 3
W. E. Ward, 6
H. Werbin, 2

BEVERLY HILLS
C. M. Agress, 1
R. W. Lippman, 1
J. Marmorston, 6
H. H. Mitchell, 6
M. Prinzmetal, 3
L. E. Travis, 1
R. E. Weston, 1

BLUE LAKE
W. J. Kerr, 3R

CARMEL
R. A. Kocher, 2R

CLAREMONT
M. D. McCarthy, 1

COLTON
V. C. Bohr, 1

CORONA DEL MAR
T. L. Patterson, 1

COSTA MESA
P. M. Nelson, 5R

CULVER CITY
B. H. Ershoff, 5

DAVIS
A. L. Black, 2
E. E. Conn, 2
D. R. Cordy, 4
J. B. Enright, 6
G. Everson, 5
H. Goss, 2, 5
C. R. Grau, 5
L. L. Ingraham, 2
M. Kleiber, 1, 5
F. H. Kratzer, 5
S. A. Peoples, 3
D. W. Peterson, 5
P. P. T. Sah, 3
C. J. Stormont, 6
P. K. Stumpf, 2

DUARTE
H. R. Bierman, 3
J. T. Holden, 2
R. Kinosita, 4
E. Roberts, 2
G. Rouser, 2
R. S. Schweet, 2
D. G. Simonsen, 2

GOLETA
J. E. Cushing, 6

GRIDLEY
W. S. Lawrence, 3

HILLSBOROUGH
J. H. Comroe, Jr., 1, 3

LA JOLLA
G. R. Bartlett, 2
H. I. Nakada, 2
N. W. Rakestraw, 2
P. F. Scholander, 1
A. N. Wick, 2

LOMA LINDA
C. R. Collier, 1
J. E. Thomas, 1, 3

LONG BEACH
K. Fink, 2

LOS ALTOS
P. M. Dawson, 1R

LOS ANGELES
J. M. Adams, 4
W. R. Adey, 1
F. Aladjem, 6
N. Alexander, 1
R. B. Alfin-Slater, 2, 5
D. E. Atkinson, 2
W. H. Bachrach, 1
N. Baker, 2
L. A. Bavetta, 5
S. A. Bencosme, 4
L. R. Bennett, 4
D. H. Blankenhorn, 4
T. H. Bullock, 1
M. N. Camien, 2
M. E. Carsten, 2
L. A. Chambers, 1, 6
A. L. S. Cheng, 5
W. G. Clark, 1, 3
C. D. Clemente, 1
F. Crescitelli, 1
H. A. Davis, 4
J. B. Dillon, 3
W. Drell, 2
D. R. Drury, 1
M. S. Dunn, 2
E. Eldred, 1
G. A. Emerson, 5
J. O. Erickson, 6
J. Field, II, 1
J. B. Field, 2
R. M. Fink, 2
N. B. Friedman, 4
E. Geiger, 5
R. George, 3
O. J. Golub, 6
I. Gordon, 6
P. O. Greeley, 1
J. D. Green, 1

CALIFORNIA – COLORADO

W. H. Griffith, 2, 5
M. I. Grossman, 1
T. J. Haley, 3
V. E. Hall, 1
J. E. Hyde, 1
C. Hyman, 1
D. T. Imagawa, 4
T. L. Jahn, 1
D. J. Jenden, 3
B. M. Kagan, 5
J. Katz, 2
E. K. Killam, 3
K. F. Killam, Jr., 3
C. R. Kleeman, 1
P. Kotin, 4
B. G. Lamson, 4
H. Latta, 4
R. L. Libby, 6
D. B. Lindsley, 1
C. G. Loosli, 4R, 6
N. S. MacDonald, 2
S. C. Madden, 4
H. W. Magoun, 1
J. V. Maloney, Jr., 1
N. S. R. Maluf, 1
W. Marx, 2
M. H. Maxwell, 1
F. W. McKee, 4
R. W. McKee, 2
J. W. Mehl, 2
W. F. H. M. Mommaerts, 1, 2
L. F. Morehouse, 1
M. Morehouse, 2
H. L. Motley, 1
E. L. Nelson, 4
P. Ortega, 4
C. F. Pait, 6
J. W. Palmer, 6
H. E. Pearson, 6
J. G. Pierce, 2
J. J. Quilligan, Jr., 6
I. Rappaport, 6
A. F. Rasmussen, 6
B. O. Raulston, 3R
P. E. Rekers, 4
R. F. Riley, 3
S. Roberts, 1, 2
J. F. Ross, 4
P. F. Salisbury, 1
P. Saltman, 2
P. R. Saunders, 3
C. H. Sawyer, 1
F. H. Scharles, 5
W. A. Selle, 1
A. L. Sellers, 1
M. W. Sezaydarian, 1
D. H. Simmons, 1
R. E. Smith, 1
J. E. Snoke, 2
S. S. Sobin, 1
R. R. Sonnenschein, 1
S. Soskin, 1, 5
M. E. Swendseid, 2
C. M. Szego, 1
D. B. Taylor, 3
C. A. Terzuolo, 1
R. D. Tschirgi, 1
R. C. Ursillo, 3
M. Verzeano, 1
D. W. Visser, 2
R. L. Walford, 4
R. Ward, 6
A. G. Ware, 2
F. W. Weymouth, 1R
J. B. Wolfe, 2
A. Wright, 4
C. L. Yuile, 4
I. Zabin, 2
L. J. Zeldis, 4

LOS GATOS
A. R. Lamb, 2R, 5

MILL VALLEY
J. R. Geary, Jr., 4
E. Oppenheimer, 3

MODESTO
H. J. Almquist, 2, 5

MONTEBELLO
H. C. Struck, 1, 3

MONTEREY
G. H. Marmont, 1

NAPA
G. W. Clark, 2R

NORTHRIDGE
B. B. Brown, 3
G. E. Cronheim, 3
G. L. Maison, 1, 3
J. R. Poppen, 1
L. W. Roth, 1, 3
J. W. Stutzman, 1, 3

A. Surtshin, 1

OAKLAND
J. E. Bacher, 2
L. W. Kinsell, 5
I. McQuarrie, 1, 4, 5R
R. J. Parsons, 4

OLIVE VIEW
M. C. D'Amour, 1

ORANGE
G. F. Warner, 1

ORINDA
J. H. Lawrence, 1

PACIFIC GROVE
A. R. Moore, 1R

PACIFIC PALISADES
J. F. Mead, 2

PALO ALTO
P. A. Cavelti, 6
B. H. J. Hofstee, 2
W. Manwaring, 4, 6
O. H. Robertson, 4R
A. M. Snell, 4

PASADENA
G. A. Allen, 1, 3
J. Bonner, 2
H. Borsook, 2
D. H. Campbell, 6
J. W. Dubnoff, 2
J. S. Garvey, 6
N. H. Horowitz, 2
K. Ishizaka, 6
G. Keighley, 1
J. B. Loefer, 1
H. K. Mitchell, 2
C. G. Niemann, 2
R. D. Owen, 6
R. W. Porter, 1
L. B. Rockland, 2

R. L. Sinsheimer, 2
R. W. Sperry, 1
C. H. Thienes, 3
A. Tyler, 6
A. van Harreveld, 1
C. A. G. Wiersma, 1
L. Zechmeister, 2

REDLANDS
C. Gruber, 1R, 3R

REDWOOD CITY
E. L. Durrum, 2
L. R. Melcher, 6

RIDGECREST
E. M. MacKay, 1

RIVERSIDE
R. W. Ridley, 3

SAN BRUNO
J. S. Thatcher, 1

SAN CARLOS
C. Chatfield, 5
F. H. Quimby, 1

SAN DIEGO
H. A. Ball, 4
B. F. Stimmel, 2

SAN FRANCISCO
T. K. Adler, 3
F. W. Allen, 2
E. L. Alpen, 1
H. H. Anderson, 3
J. S. Arnold, 4
A. R. Behnke, 1
L. L. Bennett, 1
G. R. Biskind, 4
A. L. Bloomfield, 3, 4
W. L. Bostick, 4
R. W. Brauer, 1, 3
E. Brown, 1
T. N. Burbridge, 3
S. O. Byers, 1
J. L. Carr, 4
Y. C. Chin, 3
L. J. Cole, 1
A. J. Cox, Jr., 4
S. C. Cullen, 3
A. K. Davis, 1
F. De Eds, 2, 3
R. L. Driver, 1, 3
E. L. Duggan, 2
I. S. Edelman, 1
J. J. Eiler, 2
H. W. Elliott, 3
C. Entenman, 1
R. M. Featherstone, 3
N. E. Freeman, 1
H. B. Friedgood, 1
M. Friedman, 1
W. F. Ganong, 1
Q. Geiman, 4
D. M. Greenberg, 2
L. D. Greenberg, 2
H. A. Harper, 2
C. H. Hine, 3
E. Jawetz, 6
S. M. Kalman, 3
A. Kaplan, 1
H. S. Kaplan, 4
R. H. Kellogg, 1
D. J. Kimeldorf, 1
H. I. Kohn, 1
E. Kun, 2, 3
A. E. Lewis, 1
B. Libet, 1
S. Lindsay, 4
M. E. Marsh, 1, 5
S. R. Mettler, 4
K. F. Meyer, 4R, 6
F. H. Meyers, 3
H. D. Moon, 4
H. W. Newman, 3
E. W. Page, 1
C. F. Piel, 4
H. J. Ralston, 1
L. A. Rantz, 3
L. J. Rather, 4
W. O. Reinhardt, 1
J. A. Rider, 3
K. Roberts, 1
R. H. Rosenman, 1
M. Rothstein, 2
D. A. Rytand, 3
J. J. Sampson, 1
K. G. Scott, 3
J. W. Severinghaus, 1
M. S. Silverman, 6
V. C. Sutherland, 3
M. N. Swift, 1
H. Tarver, 2
E. L. Way, 3

APO, FPO, SAN FRANCISCO
J. A. Morris, 6
E. T. Nishimura, 4
R. A. Phillips, 1
B. H. Schneider, 5

SAN JOSE
J. H. Sirota, 1

SANTA BARBARA
F. E. Bischoff, 2, 5
E. Gellhorn, 1
A. E. Koehler, 2
M. Sahyun, 2
I. Sandiford, 2R, 5R

SANTA MONICA
R. O. Bauer, 3
O. L. Huddleston, 1
J. A. Orcutt, 3
J. Tilt, 5R

STANFORD
J. P. Baumberger, 1R
J. M. Crismon, 1
W. C. Cutting, 3
R. H. Dreisbach, 3
G. A. Feigen, 1
C. S. French, 2
F. A. Fuhrman, 1
A. Furst, 3
A. Goldstein, 3
R. Grant, 1
H. S. Loring, 2
J. M. Luck, 2
S. Raffel, 6
E. Roboz, 2
E. W. Schultz, 4, 6R
C. E. Schwerdt, 2
P. E. Smith, 1R
R. E. Swain, 2R
C. Yanofsky, 2

TWENTY-NINE PALMS
R. R. Mellon, 6R

VAN NUYS
E. M. Gal, 2
J. F. Nyc, 2

WEST LOS ANGELES
A. Hemingway, 1

COLORADO

BOULDER
B. M. Tolbert, 2

COLORADO SPRINGS
H. Bourquin, 1
C. R. Schmidt, 1

DENVER
A. Abrams, 2
T. H. Allen, 1
B. O. Barnes, 1

COLORADO – CONNECTICUT – DELAWARE – DISTRICT OF COLUMBIA

M. H. Block, 4
S. C. Bukantz, 6
F. A. Cajori, 2, 5
J. R. Cann, 6
J. L. Chapin, 1
H. J. Corper, 2R
R. W. Cowgill, 2
F. E. D'Amour, 1
W. B. Draper, 3
G. F. Filley, 1
T. E. Friedemann, 2, 5
W. R. Frisell, 2
J. W. Gersten, 1
R. J. Glaser, 4
R. M. Hill, 2
J. H. Holmes, 1
S. Katsh, 1
C. H. Kempe, 6
H. F. Kraybill, 5
G. K. Lewis, 5
R. C. Lewis, 2R, 5R
C. A. Manske, 1
C. G. Mackenzie, 2, 5
J. B. Mackenzie, 2
W. P. McCann, 3
J. B. McNaught, 4
G. Meiklejohn, 6
A. V. Montgomery, Jr., 1
R. M. Mulligan, 4
E. B. Pratt, 1
T. T. Puck, 6
E. B. Reeve, 1
D. L. Smith, 3
A. Staub, 2
R. Thompson, 6
R. W. Virtue, 2
K. N. Von Kaulla, 1
R. W. Whitehead, 1, 3

FORT COLLINS
F. X. Gassner, 1

STEAMBOAT SPRINGS
E. S. Fetcher, 1

CONNECTICUT

DANBURY
E. Woll, 4

ELLINGTON
F. H. Bird, 5

GREENWICH
A. A. Albanese, 2
W. F. Loomis, 2

GUILFORD
C. S. Leonard, 3

HARTFORD
S. J. Martin, 1
K. H. Pribram, 1

MADISON
R. H. Green, 6

MANCHESTER
S. E. Pond, 1R

MIDDLETOWN
R. A. Gortner, Jr., 5

NEW BRITAIN
H. E. Munsell, 5
P. D. Rosahn, 4

NEW HAVEN
W. H. Adolph, 2R, 5
R. J. Anderson, 2
G. A. Baitsell, 1R
J. J. Barboriak, 5
D. H. Barron, 1
W. Bergmann, 2
F. L. Black, 6
C. I. Bliss, 3
E. J. Boell, 1
D. M. Bonner, 2
E. S. Canellakis, 2
C. E. Carter, 2
H. G. Cassidy, 2
H. T. Clarke, 2
G. R. Cowgill, 1R, 2, 5
J. M. R. Delgado, 1
J. English, Jr., 2
J. S. Fruton, 2
J. F. Fulton, 1
A. W. Galston, 2
N. J. Giarman, 3
J. P. Green, 3
H. S. N. Greene, 4
H. A. Harbury, 2
R. G. Harrison, 1R
L. P. Herrington, 1
D. I. Hitchcock, 2
D. Hooker, 1R
D. M. Horstmann, 6
G. D. Hsiung, 6
R. B. Hubbell, 2R, 5R
D. L. Kline, 1
W. A. Krehl, 2, 5
H. Lamport, 1
A. B. Lerner, 2
C. N. H. Long, 1, 2
E. B. Man, 2
L. H. Nahum, 1
J. S. Nicholas, 1
J. R. Paul, 4R, 6
W. H. Prusoff, 2
M. Reissig, 6
F. M. Richards, 2
I. A. Rose, 2
S. Simmonds, 2
M. V. Simpson, 2
S. J. Singer, 2
L. Stark, 1
R. M. Taylor, 6
H. P. Treffers, 6
G. van Wagenen, 1
J. T. Velardo, 1
H. B. Vickery, 2
W. Virhniac, 2
A. D. Welch, 2, 3
G. R. Wyatt, 2
I. Zelitch, 2

NEW LONDON
W. R. Miles, 1
K. E. Schaefer, 1

NEWTOWN
C. V. Bailey, 2R, 5R

NIANTIC
F. L. Dey, 1

NORTH HAVEN
E. Sachs, 1R

RIDGEFIELD
J. H. Heller, 1
M. C. Pease, 6R

ROCKY HILL
W. T. Liberson, 1

ROWAYTON
J. M. Rogoff, 1R, 3R

STAMFORD
L. Atkin, 2, 5
M. R. Thompson, 3

STORRS
E. G. Boettiger, 1
H. D. Eaton, 5
L. D. Matterson, 5

TOLLAND
W. E. Anderson, 2R, 5

WATERBURY
S. J. Liao, 6

WEST HAVEN
D. L. Buchanan, 2

WEST HARTFORD
O. R. Benham, 6
H. F. Pierce, 1

WINDSOR
H. L. Campbell, 5R

WOODBURY
J. B. Watson, 1

DELAWARE

DOVER
O. J. Pollak, 4

NEWARK
L. Brouha, 1
E. P. Radford, Jr., 1
R. R. Ronkin, 1
H. R. Rosenberg, 2, 5
G. W. H. Schepers, 4
T. R. Wood, 2
J. A. Zapp, Jr., 1

REHOBOTH
D. Marine, 1R, 4R

WILMINGTON
J. P. Frawley, 3
P. B. Hamilton, 2
M. E. Maxfield, 1
J. Waddell, 2, 5
A. T. Wilson, 6

DISTRICT OF COLUMBIA

G. Adams, 5
E. M. Anderson, 1
H. G. Armstrong, 1R
W. M. Ashby, 6
H. Bacchus, 1
S. Bayne-Jones, 4R, 6R
E. L. Becker, 6
A. S. Benenson, 6
D. F. Bogdanski, 3
W. M. Booker, 1, 3
H. A. Braun, 3
C. R. Brewer, 2
E. L. Buescher, 6
R. K. Cannan, 2
L. Carmichael, 1
H. I. Chinn, 1, 3
R. W. Clarke, 1
W. V. Consolazio, 2
G. M. Coons, 5
E. J. Coulson, 6
J. M. Curtis, 2
C. Davison, 3
H. M. Dixon, 4
D. D. Donahue, 3
J. H. Draize, 3
W. R. Duryee, 1
G. Edsall, 6
J. G. Edwards, 1
J. J. Elkes, 3
D. L. Espe, 1
J. F. Fazekas, 3
W. H. Feldman, 4
O. Felsenfeld, 6
O. G. Fitzhugh, 3
R. H. Follis, Jr., 4
T. D. Fontaine, 2
S. B. Formal, 6
F. M. Forster, 1
E. D. Freis, 3
F. Friedberg, 2
L. Friedman, 5
M. H. Friedman, 1
H. S. Fuller, 6
A. P. Gagge, 1
S. Gaines, 6
R. Galambos, 1
A. D. Glinos, 1
E. W. Goodpasture, 4
R. L. Grant, 2
D. E. Gregg, 1
A. Griffin, 6
F. E. Hahn, 2
M. Harris, 2
W. M. Hart, 1
M. L. Hathaway, 5
E. W. Hawthorne, 1
H. W. Hays, 3
W. C. Hess, 2
A. W. Hetherington, 1
E. M. Hewston, 5
C. A. M. Hogben, 1, 3
S. R. Hoover, 2
M. J. Hom, 2
P. E. Howe, 1R, 2R, 5R
G. W. Irving, Jr., 2
J. L. Johnson, 1
P. E. Johnson, 5
H. T. Karsner, 4R
O. L. Kline, 5
T. Koppanyi, 1, 3
H. A. Kuhn, 3
E. P. Laug, 2, 3
L. G. Lederer, 1
C. E. Leese, 1
A. J. Lehman, 3
R. M. Leverton, 5
L. Levin, 2
E. W. Ligon, Jr., 3
G. D. Maengwyn-Davies, 2
H. J. Magnuson, 4
H. G. Mandel, 2, 3
L. M. Marshall, 2
A. W. Martin, Jr., 1
R. P. Mason, 6
F. D. Maurer, 6
C. F. Morgan, 1, 3
E. Z. Moyer, 5
L. H. Muschel, 6
G. G. Nahas, 1
E. M. Nelson, 2, 5
L. M. Odland, 5
B. J. Olson, 6
L. W. Parr, 4
W. L. Patterson, 2
J. D. Perkinson, Jr., 2
S. N. Pradhan, 3
E. R. Ramey, 1
E. M. Renkin, 1
M. L. Robbins, 6
J. H. Roe, 2, 5
J. C. Rose, 1
G. B. Roth, 1R, 3R
J. H. Rust, 4
G. E. Schreiner, 1
A. Seidell, 2R
J. E. Smadel, 4, 6
H. F. Smetana, 4
E. R. B. Smith, 2

DISTRICT OF COLUMBIA – FLORIDA – GEORGIA – HAWAII – IDAHO – ILLINOIS

P. K. Smith, 2, 3
R. G. Smith, 3
S. L. Smith, 5R
C. R. Spealman, 1
H. K. Stiebeling, 5
J. W. Still, 1
M. X. Sullivan, 2R
W. D. Tigertt, 6
R. S. Tipson, 2
C. D. Tolle, 5
F. M. Townsend, 4
C. R. Treadwell, 2, 5
C. Voegtlin, 1R, 2R, 3R
L. Voris, 5
B. J. Vos, 3
H. G. Wagner, 1
J. R. Weisiger, 2
H. Welch, 6
F. H. Wiley, 2
V. A. Wilkerson, 2
M. C. Winternitz, 4R
A. V. Wolf, 1
M. Womack, 5
J. B. Youmans, 5

FLORIDA

CORAL GABLES
E. L. Chambers, 1
W. B. Dandliker, 2
W. B. Deichmann, 3
L. S. Dietrich, 2
J. C. Finerty, 1
W. C. Grant, 1
E. Larson, 1, 3
G. T. Lewis, 2
V. H. Moon, 4R
D. M. Morris, 1
W. G. Moss, 1
J. L. Radomski, 3
G. C. Ring, 1
M. M. Sigel, 6
R. I. H. Wang, 3
A. K. Weiss, 1

DAYTONA BEACH
N. R. Blatherwick, 1R, 2R, 5R

FORT LAUDERDALE
A. M. Hjort, 2R

FORT MYERS
J. A. E. Eyster, 1R, 3R

FORT PIERCE
M. T. Hanke, 2

GAINESVILLE
C. F. Crampton, 2
T. J. Cunha, 5
G. K. Davis, 2, 5
M. J. Fregly, 1
A. L. Koch, 2
T. H. Maren, 3
S. P. Martin, 6
J. A. Olson, 2
A. B. Otis, 1
F. W. Putnam, 2
R. L. Shirley, 5
E. Suter, 6
H. D. Wallace, 5
E. R. Woodward, 1
E. B. Wright, 1

JACKSONVILLE
A. G. Foraker, 4

MIAMI
W. A. D. Anderson, 4
D. H. Cook, 2R
D. B. Gordon, 1
[illegible]
R. Jones, Jr., 3
C. E. Lane, 1
J. B. Miale, 4
L. G. Rowntree, 1R, 2R, 3R
F. G. K. Savard, 2

MIAMI BEACH
N. Glickman, 1
P. B. Hawk, 1, 2
P. Samet, 1
M. L. Weil, 6

MIAMI SHORES
C. A. Cary, 2R

ORANGE PARK
K. S. Lashley, 1R

ORLANDO
R. M. Waters, 3R

PENSACOLA
H. W. Ades, 1
A. Graybiel, 1

PLANT CITY
O. Riddle, 1R

POMPANO BEACH
W. W. Oliver, 4R

SARASOTA
A. P. Locke, 6R
E. U. Still, 1

ST. PETERSBURG
J. W. Jobling, 4R
A. H. Lawton, 1, 3

TALLAHASSEE
L. M. Beidler, 1
K. Dittmer, 2
D. M. Easton, 1
S. W. Fox, 2
E. Frieden, 2
M. R. Sandels, 5

TAVERNIER
H. W. Haggard, 1R, 2

WEST PALM BEACH
W. H. Eddy, 2R, 5R

WINTER PARK
G. R. Crisler, 1

GEORGIA

ATHENS
M. Speirs, 5

ATLANTA
G. Bachmann, 1R
J. A. Bain, 3
F. Binkley, 2
J. H. Birnie, 1
G. A. Brecher, 1
J. H. U. Brown, 1
M. D. Cotzen, 3
F. W. Fales, 2
W. F. Friedewald, 4
J. T. Godwin, 4
S. W. Gray, 1
J. Haldi, 1
M. Hines, 1
E. N. Jenney, 3
E. Juni, 2
F. Lanni, 6
D. S. Martin, 6
S. E. Mayer, 3
J. A. Miller, Jr., 1
N. C. Moran, 3
E. Papageorge, 2
C. C. Pfeiffer, 3
A. P. Richardson, 3
J. A. Russell, 1
J. E. Steinhaus, 3
P. A. Stewart, 1
M. Tager, 6
A. E. Wilhelmi, 2
W. Wynn, 1

AUGUSTA
R. P. Ahlquist, 1, 3
L. Allen, 1
A. P. Briggs, 2R
P. Dow, 1
W. K. Hall, 2
W. F. Hamilton, 1
E. C. Jungck, 1
J. W. Remington, 1
S. A. Singal, 2, 5
V. P. Sydenstricker, 5
C.-S. Wright, 6

CHAMBLEE
A. B. Eschenbrenner, 4
I. G. Kagan, 6
S. S. Kalter, 6

COLUMBUS
W. J. Tomlinson, 4

GREENVILLE
G. C. Knowlton, 1
L. Arnold, 4, 6

SAVANNAH
W. J. Hayes, Jr., 3
R. F. Witter, 2

HAWAII

HONOLULU
G. O. Burr, 2, 5
W. A. Gortner, 2
C. D. Miller, 5

IDAHO

BOISE
E. Woods, 2R, 5R

MOSCOW
T. B. Keith, 5
A. C. Wiese, 5

ILLINOIS

CARBONDALE
H. M. Kaplan, 1
I. L. Shechmeister, 6

CHAMPAIGN
O. S. Walters, 1

CHICAGO
L. G. Abood, 1
W. R. Adams, 1
S. G. A. Alivisatos, 2
J. G. Allen, 1
S. C. Allen, 1
W. C. Alvarez, 1R, 3R
A. S. Alving, 1
R. H. Andresen, 4
H. S. Anker, 2
J. H. Annegers, 1
A. Arkin, 1R, 3R
P. Bailey, 1
H. C. Batson, 6
G. A. Bennett, 4
O. Bergeim, 1R, 2R
F. C. Bing, 2, 5
S. B. Binkley, 2
B. B. Blivaiss, 1
W. Bloom, 4
S. L. Bonting, 2
W. B. Bradley, 1
N. R. Brewer, 1
M. C. H. Brookes, 5
A. L. Brown, Jr., 4
P. C. Bucy, 1
A. M. Budy, 3
M. V. Buell, 2
W. Burrows, 6
H. Busch, 2, 3
D. H. Calloway, 5
N. O. Calloway, 3
W. J. R. Camp, 3
P. R. Cannon, 4, 6
H. R. Catchpole, 1
J. J. Ceithaml, 2
K. L. Chow, 1
J. A. Cifonelli, 2
C. Cohn, 1
D. L. Cook, 3
T. B. Coolidge, 2
E. Da Costa, 1, 5
C. W. Darrow, 1
W. Dasler, 5
I. Davidsohn, 4, 6
M. E. Davis, 3
O. F. Davis, 3
F. del Greco, 1
R. T. Dillon, 2
A. Dorfman, 2
J. R. Doty, 2
J. Doull, 3
R. M. Dowben, 1
W. L. Doyle, 1
C. A. Dragstedt, 1, 3
L. R. Dragstedt, 1
V. A. Drill, 1, 3
K. P. Dubois, 3
E. Eagle, 1, 3
D. P. Earle, Jr., 1
L. Eichelberger, 2
E. A. Evans, Jr., 2
E. B. Farnsworth, 3
R. J. Feinberg, 6
S. M. Feinberg, 6
F. W. Fitch, 4
P. P. Foa, 1
L. S. Fosdick, 2
S. Freeman, 1
J. C. Fritz, 5
N. W. Fugo, 3
H. Gaffron, 2
M. Gaudino, 1
E. M. K. Geiling, 1R, 2R, 3
I. Gersh, 4
F. A. Gibbs, 1
A. R. Goldfarb, 2
M. S. Goldstein, 1
E. Goldwasser, 2
A. S. Gordon, 1
C. E. Graham, 2, 5
E. R. Grant, 1
J. S. Gray, 1
R. Greenberg, 1
H. Greengard, 1
F. S. Grodins, 1
E. L. Gustus, 2
L. Gyemek, 3
W. C. Halstead, 1
W. E. Hambourger, 3

ILLINOIS – INDIANA

B. L. K. Hamilton, 2
C. W. Hammond, 4
D. Hamre, 6
M. E. Hanke, 2
I. Harary, 2
D. L. Harris, 2
S. C. Harris, 1, 3
R. W. Harrison, 6
G. Hass, 4
O. Hepler, 4
S. W. Hier, 2
W. T. Hill, 4
W. S. Hoffman, 2
B. N. Horwitt, 2
C. B. Huggins, 1
J. O. Hutchens, 1
R. C. Ingraham, 1
R. Isaacs, 4
A. C. Ivy, 1, 5
E. Jacobson, 1
L. O. Jacobson, 4
F. L. Jennings, 4
R. B. Jennings, 4
R. K. Jennings, 6
E. V. Jensen, 2
N. R. Joseph, 2
F. T. Jung, 1
C. M. Kagawa, 3
A. G. Karczmar, 3
L. N. Katz, 1
E. P. Kennedy, 2
A. T. Kenyon, 3
A. C. Keyl, 3
E. R. Kirch, 2
N. Kleitman, 1
H. Kluver, 1
V. L. Koenig, 2
A. J. Kosman, 1
L. M. Kozloff, 2
M. E. Krahl, 2, 3
C. A. Krakower, 4
T. H. Kritchevsky, 2
M. M. Kunde, 1R
A. C. Lane, 1
A. Lein, 1
R. Levine, 1
S. A. Levinson, 4
J. H. Lewis, 4
M. V. L'Heureux, 2
M. A. Lipton, 2
H. Lisco, 4
H. E. Longenecker, 2, 5
A. A. Luisada, 1
D. A. MacFadyen, 2
M. B. Mathews, 2
H. J. McDonald, 2
F. C. McLean, 1, 2R, 3R
J. B. Melchior, 2
C. P. Miller, 4, 6
G. H. Miller, 3
M. Morse, 2
J. W. Moulder, 2
H. Necheles, 1
W. D. Neff, 1
L. B. Nice, 1
J. L. Nickerson, 1
W. C. North, 3
Y. T. Oester, 3
G. T. Okita, 3
A. Omachi, 1
R. G. Page, 3
C. N. Peiss, 1
J. F. Perkins, Jr., 1
R. Pick, 1
C. L. Pirani, 4
C. D. Proctor, 3
C. B. Puestow, 1
T. N. Pullman, 1
N. S. Radin, 2
M. E. Rafelson, Jr., 2
W. C. Randall, 1
R. E. Ranney, 1
S. B. Raska, 1
A. L. Raymond, 2
S. R. M. Reynolds, 1
R. Rhines, 1
H. T. Ricketts, 1
B. Riegel, 2
K. C. Robbins, 6
H. E. Robinson, 5
S. R. Rosenthal, 1, 4
L. J. Roth, 3
D. A. Rowley, 4
B. B. Rubenstein, 1
A. H. Ryan, 1
O. Saphir, 4
S. Schiller, 2
F. H. Schultz, Jr., 3
N. B. Schwartz, 1
B. S. Schweigert, 2, 5
H. Schwerma, 1
H. J. Shaughnessy, 6
T. Sherrod, 3
P. Shubik, 4
L. P. Simonds, 4
A. E. Smith, 3
C. M. Smith, 3
J. A. Smith, 1
R. S. Snider, 1
B. H. Spargo, 4
H. Spector, 5
J. Stamler, 1
F. Steigmann, 3
H. B. Steinbach, 1
P. E. Steiner, 4
A. H. Steinhaus, 1
R. C. Stepto, 4
K. Stern, 4, 6
G. H. Stollerman, 6
R. T. Stormont, 3
W. W. Swanson, 2
P. Talalay, 2
L. G. Taliaferro, 6
W. H. Taliaferro, 6
D. W. Talmage, 6
A. Tannenbaum, 4
C. B. Taylor, 1, 4
J. M. Tobias, 1
J. E. P. Toman, 1, 3
K. R. W. Unna, 1, 3, 5
C. G. Van Arman, 3
B. Vennesland, 2
W. B. Wartman, 4
S. B. Weiss, 2
J. A. Wells, 3
P. L. White, 5
W. F. White, 2
W. V. Whitehorn, 1
H. G. Williams-Ashman, 2
M. B. Williamson, 2
L. C. Winter, 3
R. J. Winzler, 2
R. W. Wissler, 4, 6
G. P. Youmans, 6
E. A. Zeller, 2
J. Zichis, 6
R. E. Zirkle, 1

CHICAGO HEIGHTS
J. E. Bourque, 1

DANVILLE
J. Blumenstock, 1

DECATUR
T. B. O'Dell, 3

ELGIN
M. K. Horwitt, 2, 5

ELMHURST
J. P. Marbarger, 1

EVANSTON
G. H. Berryman, 1
F. A. Brown, Jr., 1
J. I. Farrell, 1
I. M. Klotz, 2
L. Lorand, 1, 2
H. A. McGuigan, 1R, 2R, 3R

GALESBURG
E. Costa, 3
H. E. Himwich, 1, 3
W. A. Himwich, 1
J. R. Smythies, 3

GLENVIEW
D. Nelson, 1

HIGHLAND PARK
W. C. Buchbinder, 1
J. H. Last, 3

HINES
R. Schrek, 4

HINSDALE
M. J. Schiffrin, 1

JOLIET
B. Mortimer, 1

KANKAKEE
N. Ercoli, 3

LEMONT
A. M. Brues, 1
R. N. Feinstein, 2
M. P. Finkel, 4
A. K. Laird, 4
A. Lindenbaum, 2
L. S. Lombard, 4
W. P. Norris, 2
H. M. Patt, 1
F. Schlenk, 2
J. Schubert, 2
D. E. Smith, 1
R. W. Swick, 2
T. N. Tahmisian, 1
J. F. Thomson, 1

LIBERTYVILLE
J. E. Hunter, 5

MAYWOOD
S. E. Owen, 1
E. G. Weir, 1

MORTON GROVE
H. L. Fevold, 2
R. M. Gesler, 3
H. K. Iwamoto, 3

MOUNT CARROLL
F. J. Mullin, 1

MOUNT PROSPECT
F. J. Haddy, 1

NORTH CHICAGO
G. M. Everett, 3
D. V. Frost, 2, 5
K. Hwang, 3
E. T. Kimura, 3
D. W. MacCorquodale, 2
F. C. McIntire, 2
A. E. Osterberg, 2
R. K. Richards, 1, 3
J. R. Schenck, 2
J. L. Schmidt, 3
J. D. Taylor, 3

OAK PARK
D. I. Abramson, 1
V. Guillemin, Jr., 1

PALOS PARK
E. E. Rice, 5

PEORIA
R. W. Jackson, 2, 5

QUINCY
J. W. Mull, 2

RIVERSIDE
M. A. Hinrichs, 1
E. E. Painter-Marcus, 1

ROCKFORD
W. G. Rohse, 1
D. R. Stewart, 1

SKOKIE
J. de la Huerga, 4
M. B. Gillis, 5
F. J. Saunders, 1

URBANA
D. E. Becker, 5
H. P. Broquist, 2
H. E. Carter, 2, 5
L. E. Chadwick, 1
M. W. Chang, 5
T. K. Cureton, Jr., 1
R. D. De Moss, 2
R. M. Forbes, 5
G. S. Fraenkel, 5
W. J. Fry, 1
I. C. Gunsalus, 2
T. S. Hamilton, 2, 5
A. B. Hoerlein, 6
B. C. Johnson, 2, 5
R. E. Johnson, 1, 2, 5
J. Kastelic, 5
F. A. Kummerow, 5
B. L. Larson, 2
S. E. Luria, 6
H. H. Mitchell, 2R, 5
C. L. Prosser, 1
W. C. Rose, 2, 5
W. J. Rutter, 2
F. Sargent, II, 1
H. M. Scott, 5
J. M. Smith, 5
S. Spiegelman, 1
F. R. Steggerda, 1
G. J. Thomas, 1
C. S. Vestling, 2
G. Wolf, 2
W. A. Wood, 2

INDIANA

BLOOMINGTON
H. G. Day, 2, 5
P. M. Harmon, 1
F. Haurowitz, 2, 6
H. R. Mahler, 2
L. S. McClung, 6
P. A. Nicoll, 1
S. Robinson, 1
H. H. Rostorfer, 1
T. J. B. Stier, 1
W. J. van Wagtendonk, 2
G. E. Wertenberger, 1

CEDAR LAKE
C. J. Farmer, 2R

DECATUR
W. W. Cravens, 5

ELKHART
L. A. Crandall, Jr., 1

INDIANA – IOWA – KANSAS – KENTUCKY – LOUISIANA

A. H. Free, 2, 5
W. H. Funderburk, 3
H. G. Glass, 3
R. K. S. Lim, 1, 3

EVANSVILLE
B. B. Clark, 3
W. M. Cox, Jr., 2, 5
B. K. Harned, 2, 3
F. G. McDonald, 2, 5
A. B. Morrison, 5
H. P. Sarett, 2, 5
A. L. Sheffner, 2
H. E. Stavely, 2
M. R. Warren, 3
J. H. Weikel, Jr., 3

FRANKLIN
R. H. K. Foster, 3

INDIANAPOLIS
B. E. Abreu, 3
R. C. Anderson, 3
M. H. Aprison, 1
J. Ashmore, 2
O. T. Bailey, 4
P. R. Beamer, 4
O. K. Behrens, 2
D. E. Bowman, 2
G. A. Bunde, 1
K. K. Chen, 1, 3
G. H. A. Clowes, 2R, 6R
H. A. Dettwiler, 6
E. H. Flynn, 2
R. B. Forney, 3
L. W. Freeman, 1
D. M. Gibson, 2
C. M. Gruber, Jr., 3
R. N. Harger, 2, 3
P. Harris, 3, 4
O. M. Helmer, 2
F. G. Henderson, 3
R. G. Herrmann, 3
J. B. Hickam, 1
H. R. Hulpieu, 3
K. G. Kohlstaedt, 1
C.-C. Lee, 1, 3
H. M. Lee, 3
T.-M. Lin, 1
J. F. Lyman, 2R, 5R
H. M. Powell, 6
M. A. Root, 3
E. E. Selkurt, 1
R. E. Shipley, 1
E. W. Shrigley, 6
I. H. Slater, 3
W. B. Sutton, 2
E. E. Swanson, 3
L. C. Weaver, 3

LAFAYETTE
B. Axelrod, 2
H. Beeve.s, 2
C. W. Carrick, 5
R. C. Corley, 2
J. F. Foster, 2
W. A. Hiestand, 1
M. Laskowski, Jr., 2
E. T. Mertz, 2, 5
T. S. Miya, 3
M. Moskowitz, 6
L. J. Mullins, 1
F. W. Quackenbush, 2, 5
R. L. Whistler, 2
M. X. Zarrow, 1

MARION
S. Anabacher, 2

MICHIGAN CITY
W. H. Olson, 1

NOTRE DAME
T. G. Ward, 6

SHELBYVILLE
V. B. Scott, 1

TERRE HAUTE
C. F. Failey, 2
K. E. Jensen, 6
W. C. Sherman, 5
J. Warren, 6

WEST LAFAYETTE
W. M. Beeson, 5
H. Koffler, 2

IOWA

AMES
L. Arnrich, 5
S. Aronoff, 2
W. D. Brewer, 5
W. Burroughs, 5
D. Catron, 5
E. S. Eppright, 5
D. French, 2
E. A. Hewitt, 1
L. M. Jones, 3
R. M. Melampy, 1
D. E. Metzler, 2
C. E. Roderuck,
F. G. Smith, 2
P. Swanson, 5
B. H. Thomas, 5
C. H. Werkman, 2

BURLINGTON
G. W. Changus, 4

DES MOINES
R. H. Beumer, 1R, 3
H. W. Larson, 2

IOWA CITY
C. P. Berg, 2, 5
J. T. Bradbury, 1
G. W. Brown, 1
H. Bull, 2
J. R. Carter, 4
A. L. Daniels, 2R, 5R
T. C. Evans, 1
G. E. Folk, Jr., 1
R. B. Gibson, 2R
E. G. Gross, 1, 2, 3
H. M. Hines, 1
J. P. Hummel, 2
C. J. Imig, 1
W. R. Ingram, 1
R. G. Janes, 1
W. S. Jeter, 6
R. E. Kallio, 2
G. Kalnitsky, 2
H. H. Keasling, 3
J. P. Long, 3
G. Marsh, 1
A. P. McKee, 6
R. Montgomery, 2
M. A. Ohlson, 5
I. H. Pierce, 3R
C. B. Pittinger, 3
J. R. Porter, 6
J. L. Routh, 2
F. W. Scamler, 4
G. Stearns, 2, 5
J. D. Thomson, 1
W. W. Tuttle, 1
E. D. Warner, 4
C. C. Wunder, 1

KANSAS

KANSAS CITY
G. L. Curran, 2
D. C. Darrow, 2
J. K. Frenkel, 4
R. E. Green, 3
S. Grisolia, 2
T. R. Hamilton, 4
R. M. Isenberger, 3
J. M. Nichols, 4
L. Peters, 3
K. R. Reissmann, 1
R. E. Stowell, 4
A. A. Werder, 4
W. C. Wescoe, 3

LAWRENCE
H. W. Barrett, 2
A. A. Benedict, 6
C. M. Downs, 6
K. Jochim, 1
C. A. Leone, 6
G. N. Loofbourrow, 1
R. C. Mills, 2
D. J. Mulford, 2
P. Newmark, 2
F. E. Samson, Jr., 1
N. P. Sherwood, 6
O. O. Stoland, 1R

MANHATTAN
L. Ascham, 5R
R. E. Clegg, 2
J. S. Hughes, 2, 5R
M. Kramer, 5
D. B. Parrish, 5
M. S. Pittman, 5R

OSAWATOMIE
B. Minz, 3

SALINA
C. J. Weber, 2

TOPEKA
C. C. Wang, 2, 5

WICHITA
N. F. Blau, 2
W. D. Graham, 3

WINFIELD
F. R. Miller, 4

KENTUCKY

FORT KNOX
J. R. Blair, 1
H. F. Jensen, 2
A. D. Keller, 1
W. F. Kocholaty, 2
C. J. Koehn, 5
G. W. Molnar, 1
U. F. Westphal, 2

LEXINGTON
W. L. Adams, 3
J. W. Archdeacon, 1
L. L. Boyarsky, 1
G. D. Buckner, 2R
A. J. Eisenman, 2
C. F. Essig, 1
H. F. Fraser, 3
H. E. Hill, 3
J. C. Humphries, 6
H. Isbell, 3
J. S. McHargue, 2R
M. Scherago, 6
A. Wikler, 3

LOUISVILLE
W. A. Brodsky, 1
W. F. Cantrell, 3
J. P. Holt, 1
K.-C. Huang, 1, 3
P. K. Knoefel, 3
H. Lawson, 1
M. M. Lipton, 6
K. P. McConnell, 2
J. C. Moore, 1
W. S. Rehm, Jr., 1
O. W. Shadle, 1
A. J. Steigman, 6
J. F. Taylor, 2
S. M. Walker, 1

PADUCAH
H. H. Fisher, 2

LOUISIANA

BATON ROUGE
S. L. Hansard, 5
L. L. Rusoff, 5
V. R. Williams, 2

MANDEVILLE
D. J. Cavanaugh, 3

NEW ORLEANS
A. M. Altschul, 2
R. Ashman, 1R
L. M. N. Bach, 1
H. Baer, 6
F. G. Brazda, 2
G. J. Buddingh, 4
E. Bueding, 2, 3
G. M. Carrera, 4
L. Churney, 1
R. A. Coulson, 2
C. E. Dunlap, 4
E. Farber, 2, 4
J. P. Fox, 6
G. A. Goldsmith, 5
M. H. Hack, 2
J. K. Hampton, Jr., 1
A. S. Harris, 1
W. H. Harris, 4
T. Hernandez, 1
R. L. Holman, 4
H. E. King, 1
W. A. Krivoy, 3
T. E. Mansour, 3
H. S. Mayerson, 1
H. C. McGill, Jr., 4
O. N. Miller, 2, 5
R. E. Reeves, 2
H. J. Saz, 2
O. Schales, 2
F. W. Schueler, 3
A. Segaloff, 4
M. F. Shaffer, 4, 6
E. Shaw, 2
T. C. Sherwood, 1
F. R. Skelton, 4
J. P. Strong, 4
L. A. Toth, 1
J. H. Trapold, 3
W. G. Unglaub, 5
W. B. Wendel, 2
A. G. C. White, 2
C. R. Womack, 4

MAINE

BAR HARBOR

J. L. Fuller, 1
J. B. Storer, 4
C. Ten Broeck, 4R

MARYLAND

ARMY CHEMICAL CENTER

A. M. Ambrose, 3
R. S. Anderson, 1
R. V. Brown, 1, 3
W. H. Chambers, 1, 5
F. N. Craig, 1
D. B. Dill, 1, 2
A. J. Daiemian, 1
C. C. Hassett, 1
R. G. Horton, 1
B. J. Jandorf, 2
K. K. Kimura, 3
B. P. McNamara, 3
H. O. Michel, 2
F. W. Oberst, 2, 3
J. F. O'Leary, 3
B. Sacktor, 2
N. K. Schaffer, 2
L. A. Sternberger, 6
W. H. Summerson, 2
C. tum Suden, 1
C. G. Wilber, 1
J. H. Wille, 1, 3

BALTIMORE

W. R. Amberson, 1
M. A. Andersch, 2
R. Andres, 1
E. C. Andrus, 1
A. M. Baetjer, 1
F. B. Bang, 4, 6
P. Bard, 1
F. W. Barnes, Jr., 2
R. F. Beers, Jr., 2
I. L. Bennett, Jr., 6
S. P. Bessman, 2
C. E. Bills, 2, 5
K. C. Blanchard, 3
D. Bodian, 6
J. H. Brewer, 6
T. C. Bruice, 2
H. H. Bryant, 3
R. M. Burgison, 3
A. M. Chesney, 4
F. P. Chinard, 2
B. F. Chow, 2, 5, 6
J. H. Clark, 1R
W. M. Clark, 2R
L. E. Cluff, 6
S. P. Colowick, 2
P. W. Davies, 1
F. P. Ferguson, 1
H. I. Firminger, 4
W. M. Firor, 1
W. H. Ganz, 1, 3
L. N. Gay, 6
G. O. Gey, 1
H. H. Gordon, 5
R. G. Gresell, 1
D. Grob, 1, 3
O. G. Harne, 1R
H. C. Harrison, 1
A. McG. Harvey, 1, 3
L. Helleman, 2
E. D. Hendley, 1
C. M. Herget, 1
R. M. Herriott, 2
E. Howard, 1
J. E. Howard, 1
H. A. Howe, 6
D. P. Jackson, 1
H. M. Kalckar, 2
A. R. Krall, 2
J. G. Krantz, Jr., 3
S. W. Kuffler, 1
M. Landowne, 1
R. G. Langdon, 2
M. G. Larrabee, 1
L. Lasagna, 3
A. L. Lehninger, 2, 5
W. E. Love, 1
D. I. Macht, 1R, 3R
J. W. Magladery, 1
E. K. Marshall, Jr., 1R, 2R, 3
J. McE. Marshall, 1
M. M. Mayer, 6
E. W. Maynert, 3
E. B. McCollum, 5
E. V. McCollum, 2R, 5
W. D. McElroy, 2
C. M. McKee, 6
M. Michaelis, 2
G. S. Mirick, 6
V. B. Mountcastle, 1
G. H. Mudge, 1, 3
A. Nason, 2
H. M. Nitowsky, 3
A. G. Osler, 6
R. H. Oster, 1
Z. Ovary, 6
A. H. Owens, Jr., 3
E. A. Park, 4
G. B. Pinchot, 2
A. D. Pollack, 4
D. F. Proctor, 1
J. F. Reback, 6
A. R. Rich, 4
C. P. Richter, 1
R. L. Riley, 1
J. E. Rose, 1
M. Rosenfeld, 3
R. S. Ross, 1
G. G. Rudolph, 1
A. San Pietro, 2
E. G. Schmidt, 2
W. W. Scott, 1
R. H. Shepard, 1
N. W. Shock, 1
D. C. Smith, 1
C. D. Snyder, 1R
B. L. Strehler, 2
S. R. Suskind, 6
G. F. Sutherland, 1
S. A. Talbot, 1
R. D. Teardall, 1
S. S. Tower, 1
E. B. Truitt, Jr., 3
H. J. Trumit, 1
R. R. Wagner, 6
A. E. Walker, 1
M. Walser, 1
K. Wasserman, 1
P. Whittlesey, 3
C. L. Wisseman, Jr., 6
A. C. Woods, 6R
K. L. Zierler, 1
L. P. Zill, 2

BELTSVILLE

M. Adams, 2
C. A. Denton, 5
L. P. Dryden, 5
N. R. Ellis, 2, 5
R. M. Fraps, 1
A. M. Hartman, 5
E. F. Johnston, 1
R. J. Lillie, 5
L. A. Moore, 5
H. Oldham, 5
M. H. Roepke, 3
J. F. Sykes, 1

BETHESDA

R. P. Akers, 1
E. C. Albritton, 1R
P. D. Altland, 1
B. N. Ames, 2
E. P. Anderson, 2
H. B. Andervont, 4
C. B. Anfinsen, Jr., 2
G. G. Ashwell, 2
J. Axelrod, 3
H. Baernstein, 2
C. G. Baker, 2
L. A. Barnes, 6
R. G. Bartlett, Jr., 1
R. W. Bates, 2
J. H. Baxter, 3
M. Belkin, 3
N. I. Berlin, 1
R. W. Berliner, 1
J. G. Bieri, 5
S. M. Birnbaum, 2
J. E. Birren, 1
S. Black, 2
B. Bloom, 2
B. R. Boone, 1
W. J. Bowen, 1
J. Bozicevich, 6
R. O. Brady, 2
J. H. Bragdon, 4
S. E. Branham, 6R
E. Braunwald, 1
G. Brecher, 4
G. M. Briggs, 5
B. B. Brodie, 2, 3
W. R. Bryan, 1, 4, 6
D. Burk, 2
J. J. Burns, 2, 3
M. S. Burstone, 4
E. R. Buskirk, 1
L. C. Butler, 5
G. L. Cantoni, 2, 3
C. J. Carr, 3
H. W. Chalkley, 1R
S. S. Chernick, 2
J. J. Christian, 4
R. W. Christie, 4
J. Cochin, 3
R. D. Coghill, 2
K. S. Cole, 1
P. Condliffe, 2
F. S. Daft, 2, 5
R. G. Daggs, 1, 5
A. J. Dalton, 4
J. D. Davidson, 3
D. J. Davis, 6
J. O. Davis, 1
R. LaV. Davis, 4
T. B. Dunn, 4
H. M. Dyer, 2, 5
H. Eagle, 2, 3R, 6
W. R. Earle, 4
B. E. Eddy, 6
N. B. Eddy, 3
F. Eisenberg, Jr., 2
F. P. Ellinger, 3
E. W. Emmart, 3
K. M. Endicott, 4
V. J. Evans, 4
E. V. Evarts, 1
J. L. Fahey, 1
M. Flavin, 2
J. E. Folk, 2
M. R. S. Fox, 5
K. Frank, 1
M. V. Freeman, 3
J. Freund, 6
W. H. Freygang, Jr., 1
M. G. F. Fuortes, 1
L. I. Goldberg, 3
A. Goldin, 1, 3
D. E. Goldman, 1
H. C. Goodman, 1
F. B. Gordon, 4
J. P. Greenstein, 2
P. M. Gullino, 4
K. Habel, 6
R. T. Habermann, 4
C. E. Hamilton, 1
S. W. Handford, 1
E. Hardenbergh, 1
W. F. Harrington, 2
E. C. Hartman, 5
F. W. Hartman, 4
V. B. Hartman, 1
J. Z. Hearon, 1
R. W. Hendler, 2
L. A. Heppel, 2
R. Hertz, 1
W. E. Heston, 4
B. Highman, 4
H. E. B. Hopps, 6
B. L. Horecker, 2
G. A. Hottle, 6
E. L. Hove, 5
R. J. Huebner, 6
W. C. Hueper, 4
C. P. Hutter, 1
H. A. Itano, 2
E. B. Jackson, 6
L. Jacobs, 6
W. B. Jakoby, 2
L. Karel, 3
S. Kaufman, 2
E. O. Keiles, 2, 5
T. J. Kennedy, Jr., 1
J. C. Keresztesy, 2, 5
S. S. Kety, 1, 3
R. K. Kielley, 2
W. W. Kielley, 2
M. W. Kies, 2
L. Kilham, 6
C. Kitzinger, 1
E. D. Korn, 2
C. Kometsky, 3
B. N. La Du, Jr., 2
M. Landy, 6
G. L. Laqueur, 4
L. W. Law, 4
I. G. Leder, 2
M. O. Lee, 1, 5
E. M. Lerner, II, 4
L. Levenbook, 1
L. Levintow, 2
H. L. Ley, Jr., 6
C.-L. Li, 1
C. P. Li, 6
R. D. Lillie, 4
J. C. Lilly, 1
R. B. Livingston, 1
R. Love, 4
R. C. MacCardle, 4
D. L. MacDonald, 2
H. Maling, 3
R. A. Malmgren, 4
L. H. Marshall, 1
M. E. Maver, 2
E. S. Maxwell, 2
F. J. McClure, 5
R. K. McDonald, 1
A. H. Mehler, 2
O. Mickelsen, 2, 5
G. B. Mider, 4
E. Mihalyi, 2
R. C. Millican, 3
D. Minard, 1
C. Mitoma, 2
H. P. Morris, 2, 5
R. Murray, 6
E. M. Nadel, 4
R. A. Olson, 1
J. Orloff, 1
E. A. Peterson, 2
K. A. Piez, 2
M. Pittman, 6

MARYLAND – MASSACHUSETTS

H. C. Pribor, 4
V. E. Price, 2
M. Rabinovitz, 2
A. S. Rabson, 4
D. P. Rall, 3
J. E. Rall, 1
W. Rall, 1
J. C. Reid, 2
M. E. Reid, [illegible]
S. M. Rosenthal, 3
K. K. Sanford, 4
S. J. Sarnoff, 1
J. P. Saunders, 3
R. E. Scantlebury, 1
A. E. Schaefer, 5
L. S. Schanker, 3
H. W. Scherp, 6
W. C. Schneider, 2
K. Schwarz, 2, 5
R. O. Scow, 1
J. Sendroy, Jr., 1, 2
J. Shack, 2
A. M. Shanes, 1, 3
J. A. Shannon, 1, 3
M. J. Shear, 2, 3
A. I. Shelokov, 6
E. Shelton, 4
M. B. Shimkin, 4
P. A. Shore, 3
L. Shuster, 2
M. Silverman, 2
A. Sjoerdsma, 3
F. Smith, 1
W. W. Smith, 1
H. A. Sober, 2
Leon Sokoloff, 4
Louis Sokoloff, 1
H. Specht, 1
S. S. Spicer, 1
E. R. Stadtman, 2
T. C. Stadtman, 2
S. N. Stein, 1
D. Steinberg, 2
H. G. Steinman, 2
DeW. Stetten, Jr., 2
M. R. Stetten, 2
H. L. Stewart, 4
E. F. Stohlman, 3
F. Stohlman, 4
S. H. Stone, 6
C. W. Tabor, 3
H. Tabor, 2, 3
I. Tasaki, 1
R. E. Taylor, 1
E. O. Titus, 2, 3
G. M. Tomkins, 2
Y. J. Topper, 2
S. Udenfriend, 2, 3
R. Van Reen, 5
W. F. Von Oettingen, 3
D. M. Watkin, 1, 5
E. C. Weinbach, 2
A. Weissbach, 2
J. White, 2
G. Z. Williams, 4
R. B. Williams, Jr., 4
H. N. Wilson, 2
W. F. Windle, 1
M. Winitz, 2
S. H. Wollman, 1
M. W. Woods, 2
W. G. Workman, 6
B. E. Wright, 2
C. I. Wright, 1, 3
I. Zipkin, 5
C. G. Zubrod, 3

CAMBRIDGE

C. T. Morgan, 1

CHEVY CHASE

C. Ajmone-Marsan, 1
D. E. Copeland, 1
D. M. Rioch, 1
O. Schreiner, 2R
J. Steinhardt, 2

CLARKSBURG

O. E. Reynolds, 1

COLLEGE PARK

G. F. Combs, 5
H. W. Schoenborn, 1
J. C. Shaw, 1

FORT DETRICK

J. B. Bateman, 1
T. O. Berge, 6
L. D. Fothergill, 6
C. A. Gleiser, 4
C. B. Thome, 2
G. G. Wright, 6
R. H. Yager, 6

FREDERICK

J. D. McCarrell, 1
M. W. Slein, 2

KENSINGTON

H. D. Bruner, 1, 3
H. Edelhoch, 2
W. H. Marshall, 1
F. R. White, 2

ROCKVILLE

T. H. Benzinger, 1

SILVER SPRING

H. G. Barott, 5R
E. L. Batchelder, 5
W. S. Gochenour, Jr., 6
B. G. King, 1
A. A. Nelson, 4
M. F. Stanton, 4

TAKOMA PARK

H. Y. C. Wong, 1

TOWSON

P. J. Crittenden, 1, 3

WALDORF

F. A. Csonka, 2

MASSACHUSETTS

AMHERST

V. C. Dewey, 2
J. O. Holmes, 2, 5
G. W. Kidder, 2
H. N. Little, 2
H. S. Mitchell, 5
J. G. Snedecor, 1
A. W. Wertz, 5

ARLINGTON

J. C. R. Licklider, 1

BOSTON

B. Alexander, 2
F. N. Allan, 4
S. B. Andrus, 4
J. Aub, 1R, 5
E. E. Baker, 6
E. G. Ball, 2
W. A. Bardawil, 4
A. C. Barger, 1
W. Bauer, 1
W. S. Beck, 2
H. K. Beecher, 1, 3
K. Benirschke, 4
G. P. Berry, 6
W. C. Boyd, 6
M. A. B. Brazier, 1
F. N. Briggs, 3
A. F. Brodie, 2
E. A. Brown, 6
N. L. R. Bucher, 1
J. P. Bunker, 3
B. S. Burke, 3
A. Canzanelli, 1
W. B. Castle, 4
B. Castleman, 4
J. Cohen, 4
A. H. Coons, 6
B. G. Covino, 1
J.M. Craig, 4
C. S. Davidson, 5
B. D. Davis, 2, 3
P. B. Dews, 3
L. Dexter, 1
L. Dienes, 6
R. P. Durbin, 1
M. D. Eaton, 6
D. Elwyn, 2
J. F. Enders, 6
L. L. Engel, 2
B. E. Ersten, 3
S. Farber, 4
J. M. Felts, 1
M. Finland, 6
W. H. Fishman, 2
G. E. Foley, 4
E. M. Follensby, 6
W. H. Forbes, 1
A. S. Freedberg, 1
D. G. Freiman, 4
E. H. Frieden, 2
M. E. Friedkin, 2
D. G. Friend, 3
S.-C. J. Fu, 2
G. P. Fulton, 1
J. Furth, 4
O. Gates, 4
J. Gergely, 2
S. N. Gershoff, 5
R. P. Geyer, 5
D. Gitlin, 6
W. T. Goodale, 1
L. Gorini, 2
R. O. Greep, 1
J. Gross, 1
R. E. Gross, 4
Z. Hadidian, 1, 3
P. B. Hagen, 3
D. D. Hagerman, 2
A. H. Handler, 4
J. H. Hanks, 6
A. B. Hastings, 1, 2, 5
J. M. Hayman, Jr., 1, 3
F. W. Haynes, 1
A. H. Hegnauer, 1
D. M. Hegsted, 5
E. Heinz, 2
E. E. Hellerstein, 4
E. Henneman, 1
A. Hertig, 4
H. H. Hiatt, 2
S. P. Hicks, 4
M. B. Hoagland, 2
F. L. Hoch, 2
R. W. Jeanloz, 2
E.P. Joslin, 5R
M. L. Karnovsky, 2
E. H. Kass, 6
E. B. Keller, 2
C. J. Kensler, 3
H. B. Kenton, 6
J. H. Kinoshita, 2
W. B. Kinter, 1
E. Knobil, 1
W. E. Knox, 2
O. Krayer, 3
J. P. Kulka, 4
E. M. Landis, 1
S. Leskowitz, 6
R. R. Levine, 3
T. H. Li, 3
F. J. Lionetti, 2
C. Liu, 6
E. R. Loew, 1, 3
R. B. Loftfield, 2
J. M. Looney, 2
F. C. Lowell, 6
M. Lubin, 3
B. R. Lutz, 1R
C. L. Maddock, 4
S. Maddock, 4
B. Magasanik, 2
G. Majno, 4
S. Malkiel, 6
G. K. Mallory, 4
E. J. Masoro, 1
J. Mayer, 1, 5
R. A. McFarland, 1
D. G. McKay, 4
J. Mead, 1
W. A. Meissner, 4
A. Meister, 2
J. P. Merrill, 1
K. Moldave, 2
H. S. Moyed, 2
P. L. Munson, 2, 3
M. M. Nothman, 3
J. P. O'Hare, 4R
J. L. Oncley, 2
J. C. Opsahl, 2
J. R. Pappenheimer, 1
E. W. Pelikan, 3
M. L. Quaife, 2, 5
F. M. Rackemann, 6
D. Rapport, 1
A. S. Relman, 1
A. E. Renold, 1
S. L. Robbins, 4
F. L. Rodkey, 2
H. F. Root, 5
A. B. Russfield, 4
K. Schmid, 2
R. Schmid, 4
G. Schmidt, 2
W. B. Schwartz, 1
J. N. Shanberge, 4
J. H. Shaw, 5
F. M. Sinex, 2
T. C. Smith, 3
F. F. Snyder, 1
J. C. Snyder, 6
A. K. Solomon, 1
S. C. Sommers, 4
F. J. Stare, 2, 5
C. F. Szittmatter, 2
D. MacN. Surgenor, 2
E. B. Taft, 4
S. J. Thannhauser, 2
G. W. Thom, 1
E. H. Tompkins, 4
U. G. Trendelenburg, 3
H. C. Trimble, 2, 5R
M. F. Trulson, 5
J. L. Tullis, 4
H. E. Umbarger, 2
B. L. Vallee, 2
C. A. Villee, Jr., 2
J. J. Vitale, 5
J. H. Wallace, 6
C. W. Walter, 4
S. Warren, 4
B. K. Watson, 6
T. H. Weller, 6
C. W. White, Jr., 3
P. D. White, 3
J. L. Whittenberger, 1
T. H. Wilson, 2
H. H. Wotiz, 2
L. C. Wyman, 1

MASSACHUSETTS – MICHIGAN

F. E. Yates, 1
A. G. Young, 3
N. Zamcheck, 4
P. Zamecnik, 2

BRIGHTON
M. Stefanini, 1, 4, 6

BROOKLINE
A. B. Astwood, 1
J. B. Daniels, 6
J. L. Gamble, 2R, 5R
H. Wu, 2

CAMBRIDGE
P. Bernfeld, 2
K. Bloch, 2
J. M. Buchanan, 2
E. S. Castle, 1
P. M. Doty, 2
J. T. Edsall, 2
M. M. Eliot, 5
C. Floyd, 6
A. Forbes, 1
B. S. Gould, 2
D. R. Griffin, 1
A. Gulick, 1R, 5R
L. P. Hager, 2
R. S. Harris, 5
F. L. Hisaw, 1
F. Homburger, 4
R. H. Hubbard, 2
J. Y. Lettvin, 1
C. P. Lyman, 1
J. Marmur, 2
J. W. Mavor, 1R
W. S. McCulloch, 1
G. A. Miller, 1
A. M. Pappenheimer, Jr., 2, 6
F. O. Schmitt, 1
I. W. Sizer, 1, 2
S. S. Stevens, 1
T.-t. Tchen, 2
G. Wald, 2
P. D. Wall, 1
J. D. Watson, 2
D. F. Waugh, 1, 2
J. H. Welsh, 1
L. G. Wesson, 2R, 3
F. H. Westheimer, 2
C. M. Williams, 1

CHESTNUT HILL
L. Rane, 6

COCHITUATE
M. G. Gray, 3

CONCORD
R. M. Ferry, 2

FITCHBURG
B. S. Walker, 2

FRAMINGHAM CENTRE
P. F. Iampietro, 1

HADLEY
E. V. Smith, 6

HINGHAM
D. L. Belding, 4

JAMAICA PLAIN
J. Ipsen, Jr., 6

LEXINGTON
C. H. Fiske, 2R
B. H. Waksman, 6

LYNN
S. E. Steiman, 1

MEDFORD
K. D. Roeder, 1
R. Rustigian, 6

MILTON
H. S. Forbes, 1R
G. P. Grabfield, 3R

MONSON
W. Hale, 1R, 3R

NATICK
D. E. Bass, 1
O. A. Bessey, 2, 5
A. F. Henschel, 1
D. H. K. Lee, 1
G. L. Miller, 2
A. H. Woodcock, 1

NEEDHAM
M. F. Crawford, 1

NEWTON
H. L. Higgins, 1, 5R

NEWTON HIGHLANDS
F. W. Maurer, 1

NEWTON UPPER FALLS
I. A. Macchi, 1

NORTHAMPTON
M. Sampson, 5

SHREWSBURY
J. R. Bergen, 1
M. C. Chang, 1
R. I. Dorfman, 2
M. Hayano, 2
O. M. Hechter, 1
E. L. Hess, 2
H. Hoagland, 1
W. P. Koella, 1
G. Pincus, 1
E. B. Romanoff, 1
H. Rosenkrantz, 2
D. Stone, 2
C. Temer, 2

SOUTHBRIDGE
O. W. Richards, 1

SOUTH HADLEY
C. Haywood, 1
J. Muus, 2

SOUTH WEYMOUTH
C. W. Duval, 4R

SPRINGFIELD
P. V. Karpovich, 1

WABAN
R. G. Hoskins, 1R
M. S. Raben, 1

WALPOLE
L. C. Fogg, 4

WALTHAM
W. P. Jencks, 2
M. E. Jones, 2
M. D. Kamen, 2
N. O. Kaplan, 2
L. Levine, 6
M. Soodak, 2

WAVERLEY
J. Folch, 2
A. Pope, 2

WELLESLEY
M. E. Collett, 1R
E. E. Jones, 4
L. P. Wilson, 1

WELLESLEY HILLS
F. H. Pratt, 1R

WEST NEWTON
F. H. McCrudden, 2R
G. F. Vawter, 4

WESTON
B. G. Ferris, Jr., 1

WESTWOOD
T. M. Carpenter, 1R, 2R, 5R

WILLIAMSTOWN
S. A. Matthews, 1

WINCHESTER
L. Varga, 2

WOODS HOLE
R. Benesch, 2
R. E. Benesch, 2
A. G. Marshak, 1
A. C. Redfield, 1R
P. Rieser, 1
A. Szent-Györgyi, 2
A. G. Szent-Györgyi, 1, 2

WORCESTER
F. Elmadjian, 1
A. P. Truant, 3

MICHIGAN

ANN ARBOR
W. W. Ackermann, 2, 6
B. L. Baker, 1
J. W. Bean, 1
I. A. Bernstein, 2
F. H. Bethell, 4
W. D. Block, 2
J. J. Blum, 1
D. F. Bohr, 1
C. R. Brassfield, 1
T. M. Brody, 3
D. E. S. Brown, 1
G. C. Brown, 6
E. J. Cafruny, 3
E. A. Carr, Jr., 3
J. P. Chandler, 2, 5
H. N. Christensen, 2
A. A. Christman, 2
K. W. Cochran, 3, 6
M. J. Coon, 2
F. M. Davenport, 6
H. W. Davenport, 1
V. D. Davenport, 1
E. F. Domino, 3
R. W. Doty, 1
H. C. Eckstein, 2
R. C. Elderfield, 2
L. H. Elwell, 1
T. Francis, Jr., 4, 6
I. B. Fritz, 1
R. W. Gerard, 1
G. R. Greenberg, 2
R. C. Hendrix, 4
A. V. Hennessy, 6
J. F. Hogg, 2
I. M. Hoobler, 2, 5
S. W. Hoobler, 1
V. Iob, 2
A. G. Johnson, 6
M. W. Johnston, 2
R. L. Kahn, 4, 6
M. Lawrence, 1
L. H. Louis, 2
M. Mason, 2
L. B. Mellett, 3
L. Miller, 2
W. J. Nungester, 6
R. L. Potter, 2
T. R. Riggs, 2
S. Roseman, 2
M. H. Seevers, 1, 3
B. F. Steele, 5
C. C. Sturgis, 4
H. H. Swain, 3
A. H. Wheeler, 6
W. S. Wilde, 1
L. A. Woods, 3

CARO
V. V. Cole, 3

DEARBORN
C. L. Schneider, 1

DETROIT
P. D. Bartlett, 2
D. H. Basinski, 2
W. T. Beher, 2
O. Bird, 2
A. C. Bratton, Jr., 3
R. A. Brown, 5
G. Chen, 3
A. J. Derbyshire, 1
P. R. Dumke, 3
F. G. Evans, 1
C. E. Frohman, 2
O. H. Gaebler, 2
E. D. Gardner, 1
E. B. Gerheim, 1, 6
A. J. Glazko, 1
A. Golden, 4
M. Goodman, 6
A. L. Grafflin, 1
R. M. Johnson, 2
S. A. Johnson, 1
C. G. Johnston, 1
E. B. Kearney, 2
V. E. Kinsey, 2
P. A. Kober, 2R
A. C. Kuyper, 2
B. M. Lewis, 1
G. A. Lo Grippo, 4, 6
W. O. Maddock, 1
D. A. McGinty, 1
I. W. McLean, Jr., 6
R. W. Monto, 4
F. A. Morin, 1
J. Nyboer, 1
A. U. Orten, 5
J. M. Orten, 2, 5
B. Pearson, 4
J. J. Pfiffner, 1, 2, 5
J. W. Rebuck, 4
D. J. Sandweiss, 1
W. H. Seegers, 1, 2
M. R. Sikov, 4
T. P. Singer, 2
A. H. Smith, 2, 5
P. G. Stansly, 2
A. R. Taylor, 6
A. J. Vorwald, 4
J. K. Weston, 4
C. V. Winder, 1, 3

EAST LANSING
B. V. Alfredson, 1
R. S. Bandurski, 2
S. H. Bartley, 1
R. U. Byerrum, 2
D. C. Cederquist, 5
W. D. Collings, 1
M. Dye, 2R, 5R
R. J. Evans, 5

MICHIGAN – MINNESOTA – MISSISSIPPI – MISSOURI

J. L. Fairley, Jr., 2
L. C. Ferguson, 6
R. G. Hansen, 2
R. M. Herbst, 2
J. A. Hoefer, 5
C. A. Hoppert, 5
C. F. Huffman, 5
R. W. Luecke, 5
W. N. Mack, 6
J. Meites, 1, 5
T. Porter, 5
E. P. Reineke, 1
H. M. Sell, 2
N. E. Tolbert, 2
C. K. Whitehair, 5
L. F. Wolterink, 1

GRAND RAPIDS
C. J. Herrick, 1R
J. D. Mann, 4

GROSSE POINTE
O. M. Gruhzit, 4

GROSSE POINTE FARMS
O. Kamm, 2R

KALAMAZOO
W. B. Bass, 3
G. C. Bond, 6
G. F. Cartland, 2
W. A. Freyburger, Jr., 3
M. E. Greig, 3
R. W. Heinle, 5
M. H. Kuizenga, 2
C. E. Meyer, 2
P. H. Seay, 3
M. J. Vander Brook, 3
F. E. Visscher, 1

LANSING
A. K. Boot, 2R
R. Y. Gottshall, 6
C. W. Muehlberger, 3

MIDLAND
M. B. Chenoweth, 3
L. C. Hendershot, 3
D. D. Irish, 3
G. N. Smith, 2

MINNESOTA

AUSTIN
R. T. Holman, 2, 5
W. O. Lundberg, 2

MINNEAPOLIS
J. T. Anderson, 5
W. D. Armstrong, 1, 2
A. B. Baker, 4
C. P. Barnum, Jr., 2
E. T. Bell, 4
W. M. Benson, 3
R. N. Bierer, 3
P. D. Boyer, 2
E. B. Brown, Jr., 1
J. Brozek, 1, 5
J. G. Brunson, 4
B. Campbell, 1
C. W. Carr, 2
W. O. Caster, 2
H. M. Cavert, 1
E. M. Cranston, 3
J. R. Dawson, Jr., 4
G. T. Evans, 1
G. Fahr, 1R, 3R
I. D. Frantz, 2
A. W. Frenkel, 2
D. Glick, 2
R. A. Good, 4, 6
F. Grande, 1, 5
H. R. Gutmann, 2
F. Halberg, 1
F. W. Hoffbauer, 4
J. A. Johnson, 1
H. Jonas, 1
C. Kennedy, 2R, 5R
A. Keys, 1, 2
J. T. King, 1
C. B. Knodt, 5
F. J. Kottke, 1
W. G. Kubicek, 1
H. C. Lichstein, 2
N. Lifson, 1
V. Lorber, 1
R. Lumry, 2
C. Martinez, 1
G. A. Matson, 6
L. O. Pilgeram, 2
E. C. Rosenow, 4R, 6R
W. F. Scherer, 6
O. H. Schmitt, 1
S. Schwartz, 2
E. Simonson, 1
L. Singer, 2
L. I. Smith, 2
W. W. Spink, 6
J. T. Syverton, 4, 6
H. L. Taylor, 1
A. P. Thal, 4
L. Tobian, Jr., 1
J. F. VanPilsum, 2
R. L. Vernier, 4
M. B. Visscher, 1
R. W. Von Korff, 2
O. H. Wangensteen, 1, 4
C. J. Watson, 2
D. W. Watson, 6
L. W. Wattenberg, 4
H. S. Wells, 1
F. W. Wittich, 6
H. N. Wright, 3
B. Zimmerman, 4

PAYNESVILLE
E. D. Brown, 1R, 3R

ROCHESTER
A. Albert, 1
S. Amberg, 1R, 2R, 3R
R. C. Bahn, 1
E. J. Baldes, 1
R. G. Bickford, 1
J. L. Bollman, 4
H. B. Burchell, 1
H. R. Butt, 5
C. F. Code, 1, 3
J. E. Edwards, 4
H. E. Essex, 1, 3R
A. Faulconer, Jr., 3
G. A. Fleisher, 2
E. V. Flock, 2
W. S. Fowler, 1
J. H. Grindlay, 1
G. A. Hallenbeck, 1
J. F. Herrick, 1
W. H. Hollinshead, 1
V. Johnson, 1
N. M. Keith, 1, 3R, 4
E. H. Lambert, 1
J. S. Lundy, 3
T. B. Magath, 1
F. C. Mann, 1R, 4R
H. L. Mason, 2
D. R. Mathieson, 6
V. R. Mattox, 2
K. N. Ogle, 1
C. A. Owen, Jr., 1, 4
M. H. Power, 2
G. M. Roth, 1
A. H. Sanford, 6R
C. Sheard, 1
H. J. C. Swan, 1
K. G. Wakim, 1, 3
R. M. Wilder, 1R, 4R, 5R
E. H. Wood, 1, 3

ST. PAUL
D. R. Briggs, 2
W. F. Geddes, 2
R. Jenness, 2
S. Kirkwood, 2
J. M. Leichsenring, 5
L. E. Liener, 2, 5
W. E. Petersen, 1
M. O. Schultze, 2, 5
A. V. Scoessler, 6
W. T. S. Thorp, 4

MISSISSIPPI

JACKSON
A. C. Guyton, 1
J. D. Hardy, 1
J. G. Hilton, 1, 3
C. C. Randall, 4, 6
J. C. Rice, 3
W. L. Williams, 1, 4

MISSOURI

COLUMBIA
B. Bisbey, 5R
H. E. Dale, 1
J. E. Flynn, 4
C. C. Guthrie, 1R, 3R
P. Hitchcock, 3
A. G. Hogan, 2R, 5
R. L. Jackson, 5
T. D. Luckey, 5
D. T. Mayer, 2
A. W. Merrick, 1
D. K. Meyer, 1
B. L. O'Dell, 2, 5
W. H. Pfander, 5
W. S. Platner, 1
J. E. Randall, 1
B. A. Westfall, 1, 3

FLORISSANT
P. L. Varney, 6

JOPLIN
R. L. Ferguson, 4

KANSAS CITY
B. W. Beadle, 2
L. L. Eisenbrandt, 3
B. K. Forscher, 4
G. C. Rau, 1

KIRKSVILLE
J. S. Denslow, 1
F. T. Dun, 1
S. F. Howell, 2
I. M. Korr, 1

ST. LOUIS
E. Adams, 2
W. M. Allen, 1
P. Berg, 2
R. M. Bethke, 2, 5
R. J. Bing, 1
G. H. Bishop, 1
H. T. Blumenthal, 4
G. O. Broun, 4
D. H. Brown, 2
H. B. Burch, 2, 5
R. M. Burton, 2
M. H. Claro, 1
S. Cohen, 2
Melvin Cohn, 2
Mildred Cohn, 2
B. Commoner, 2
C. F. Cori, 2
R. K. Crane, 2
H. Davis, 1
E. W. Dempsey, 1
E. A. Doisy, Sr., 2
E. A. Doisy, Jr., 2
H. N. Eisen, 6
W. H. Elliott, 2
J. Erlanger, 1R
M. S. Fleisher, 4, 6
A. S. Gilson, Jr., 1
S. Goldring, 1
H. T. Graham, 1, 3
D. M. Green, 1, 3
C. G. Harford, 6
J. C. Harkin, 4
W. J. Harrington, 4
A. F. Hartmann, 2
W. S. Hartroft, 4
P. Heinbecker, 1
A. B. Hertzman, 1
S.-L. Hsia, 2
F. E. Hunter, Jr., 2, 3
P. A. Katzman, 2
J. E. Kirk, 2
A. Kornberg, 2
G. L. Kreezer, 1
P. E. Lacy, 4
W. M. Landau, 1
L. Loeb, 1R, 4
O. H. Lowry, 2, 3
S. A. Luse, 4
L. J. Machlin, 5
C. V. Moore, 4, 5
C. A. Moyer, 1
X. J. Musacchia, 1
C. H. Neilson, 2
R. M. O'Neal, 4
H. Pinkerton, 4
P. W. Preisler, 2
A. W. Richardson, 1
E. Robins, 2
E. Ronzoni, 2
A. Roos, 1
T. Rosebury, 6
R. W. Schlesinger, 6
G. M. Schoepfle, 1
P. A. Shaffer, 2
R. E. Shank, 2
S. Sherry, 1
W. J. Siebert, 4
M. Silberberg, 4
R. Silberberg, 4
W. Sleator, Jr., 1
J. R. Smith, 1
M. Somogyi, 2R
D. R. Strength, 2
J. L. Strominger, 2, 3
S. A. Thayer, 2
W. A. Thomas, 4
S. F. Velick, 2
N. J. Wade, 1
Z. Wallen-Lawrence, 2
T.-Y. Wang, 2
T. E. Weichselbaum, 2
H. L. White, 1
R. D. Williams, 5

SPRINGFIELD
S. R. Johnson, 5

WEBSTER GROVES
F. E. Franke, 1

MONTANA – NEBRASKA – NEW HAMPSHIRE – NEW JERSEY – NEW MEXICO – NEW YORK

MONTANA

GREAT FALLS
E. J. Eichwald, 4

HAMILTON
C. M. Eklund, 4
D. B. Lackman, 6
C. L. Larson, 6
S. B. Salvin, 6

MISSOULA
J. J. Munoz, 6

NEBRASKA

LINCOLN
R. Borchers, 5
R. E. Feeney, 2
H. G. O. Holck, 1R, 3
R. B. Johnston, 2
W. E. Militzer, 2
D. M. Pace, 1
L. C. Payne, 1
J. H. Pazur, 2

OMAHA
A. L. Bennett, 1
A. W. Brody, 1
A. L. Dunn, 1
J. Ferguson, 1
J. R. Johnson, 1
J. Kramar, 1
V. E. Levine, 2
A. R. McIntyre, 1, 3
S. Morgulis, 1R, 2R
H. J. Phillips, 1
S. C. Samaras, 1
J. R. Schenken, 4
C. M. Wilhelmj, 1

NEW HAMPSHIRE

CONWAY
J. C. Andrews, 2R

DURHAM
A. E. Teeri, 5
P. A. Wright, 1

HANOVER
D. J. Botts, 1
F. G. Carpenter, 1
J. H. Copenhaver, 2
R. P. Forster, 1
R. E. Gosselin, 1, 3
M. F. Morales, 1
L. Noda, 2
S. M. Tenney, 1

LANCASTER
E. S. Robinson, 6

RAYMOND
G. L. Foster, 2R

WINCHESTER
O. W. Barlow, 3

NEW JERSEY

BAYONNE
D. Melnick, 2, 5

BELLEVILLE
J. F. Reinhard, 3

BERGENFIELD
C. A. Smith, 1R

BLOOMFIELD
W. M. Govier, 3
C. D. Hendley, 1
S. Irwin, 3
A. S. Meyer, 2
P. L. Perlman, 1, 3
F. E. Roth, 3
L. I. A. Tabachnick, 3
R. E. Waterman, 2
M. M. Winbury, 3

BOGOTA
E. L. Scott, 1R, 2R

BOUND BROOK
E. E. Howe, 2
S. Margolin, 3

CAMDEN
C. H. Krieger, 5
W. J. Snape, 1
D. Tourtellotte, 5

CHATHAM
J. R. Oliver, 1, 4

DUMONT
S. M. Beiser, 6

ENGLEWOOD
E. M. Vahlteich, 5

FAIR LAWN
I. B. Wilson, 2

JERSEY CITY
D. D. Bonnycastle, 3
P. J. Costa, 3
R. L. Garner, 2
S. B. Gertner, 3
H. G. Grady, 4
D. E. Hutcheon, 3
R. C. Little, 1
D. F. Opdyke, 1
S. Weiss, 2

LEONIA
R. C. Darling, 1

MADISON
E. C. Hay, 1

MAYWOOD
J. J. Oleson, 2
S. Y. P'an, 3

MONTCLAIR
R. H. Muller, 6
E. L. Severinghaus, 2, 5

MORRIS PLAINS
L. E. Arnow, 2
R. L. Kroc, 1
C. C. Scott, 3
R. A. Turner, 2

MORRISTOWN
G. H. Mangun, 2

NEWARK
J. V. Scudi, 2, 3, 5

NEW BRUNSWICK
J. B. Allison, 2, 5
A. A. Boyden, 6
W. E. Bunney, 6
J. C. Burke, 3
B. N. Craver, 3
J. D. Dutcher, 2
L. N. Ellis, 5
H. Fisher, 5
J. Fried, 2
J. F. Gardocki, 3
V. Groupe, 6
M. Heidelberger, 2R, 6
T. O. King, 3
W. L. Koerber, 6
J. O. Lampen, 2
J. H. Leathem, 1
C. R. Linegar, 3
G. Litwack, 2
W. J. Nickerson, 2
O. J. Plescia, 6
M. A. Reid, 1
B. Rubin, 3
M. Solotorovsky, 6
R. L. Squibb, 5
P. D. Sturkie, 1
M. W. Taylor, 5
W. W. Umbreit, 2
M. Upton, 1
H. J. Vogel, 2
W. W. Wainio, 2
H. S. Weiss, 1
O. Wintersteiner, 2

NEWTON
H. W. Titus, 2, 5

NORTH PLAINFIELD
C. G. Hartman, 1R

NUTLEY
J. C. Bauernfeind, 5
L. O. Randall, 2, 3
S. H. Rubin, 2, 5
W. B. Schallek, 1, 3

ORADELL
A. F. Coca, 6H

PALISADES PARK
L. B. Dotti, 1

PARK RIDGE
A. W. Moyer, 6

PLAINFIELD
K. Folkers, 2, 5

PRINCETON
F. M. Berger, 3
C. H. Bunting, 4R
A. M. Chase, 1, 2
H. Cohen, 1
E. N. Harvey, 1R, 2R
F. H. Johnson, 1
E. C. Kendall, 1, 2R, 4R
A. K. Parpart, 1
W. W. Swingle, 1

RAHWAY
L. B. Achor, 3
D. K. Bosshardt, 5
N. G. Brink, 2
I. Clark, 2
U. J. Lewis, 2
H. Molitor, 1, 3
C. W. Musbett, 4
H. J. Robinson, 3
R. W. Schayer, 1, 2
O. H. Siegmund, 3
R. H. Silber, 2
H. C. Stoerk, 4
D. M. Tennent, 2

RARITAN
W. J. Haines, 2
P. Levine, 6
H. O. Singher, 1

RIDGEWOOD
E. H. Dearborn, 3

ROSELLE PARK
G. P. Child, 3

SKILLMAN
L. Pearce, 4R

SOMERVILLE
R. P. Herwick, 3
S. Krop, 1, 3
G. Lu, 3
N. Millman, 1
W. Van Winkle, Jr., 3

SUMMIT
R. Gaunt, 1
R. A. Maxwell, 3
A. J. Plummer, 3
J. A. Schneider, 1, 3
A. Spoha, 5
R. R. Williams, 2R, 5
F. F. Yonkman, 3

SUSSEX
J. P. Mixner, 1

TEANECK
M. E. Shils, 5

WESTFIELD
G. E. Boxer, 2
M. G. Mulinos, 3
W. H. Ott, 5

WESTWOOD
L. A. Underkofler, 2

NEW MEXICO

ALBUQUERQUE
W. J. Eversole, 1
F. G. Hirsch, 1
B. B. Longwell, 2
U. C. Luft, 1
E. E. Nelson, 3R
E. A. Pinson, 1

LOS ALAMOS
I. J. U. Boone, 2
R. G. Gould, 2
W. H. Langham, 2
C. C. Lushbaugh, 4
D. F. Petersen, 3
S. Shlaer, 1

NEW YORK

ALBANY
R. S. Alexander, 1
J. Bourdillon, 2
R. Brown, 6
R. K. Brown, 2
J. M. Coffey, 6
S. M. Cohen, 6
F. C. Ferguson, Jr., 3
S. Garb, 3
A. H. Harris, 6
E. L. Hazen, 6
J. L. Hendry, 6
G. S. Kanter, 1
J. F. Kent, 6
G. F. Maley, 2
F. Maltaner, 6R

NEW YORK

J. A. Mantz, 2
M. C. Pangborn, 2
L. Rosenblum, 3
M. Shaw, 6
G. R. Sickles, 6
V. N. Tompkins, 4
H. Van Vunakis, 2
M. W. Wheeler, 6
H. C. Wiggers, 1
A. W. Wright, 4

ALFRED
H. O. Burdick, 1

ARDSLEY
E. B. Sigg, 1

BELLEROSE
A. W. Walter, 6

BRONXVILLE
G. B. Elion, 2

BROOKLYN
H. G. Albaum, 2
J. Belford, 3
M. R. Bovarnick, 2
K. L. Bowman, 6
C. McC. Brooks, 1, 3
W. D. Celmer, 2
R. H. Cheney, 3
P. F. Cranefield, Jr., 1
E. A. Eckert, 4, 6
A. H. Eggerth, 4
D. Fetter, 1
P. J. Fitzgerald, 4
R. F. Furchgott, 2, 3
C. H. Greene, 1, 2, 4
J. Gross, 1
R. M. Guttman, 1
J. B. Hamilton, 1
G. R. Hennigar, 4
G. L. Hobby, 6
B. F. Hoffman, 1
H. T. Huang, 2
C.-Y. Kao, 1
F. F. Kao, 1
K. Koizumi, 1
B. Kramer, 1, 2
D. M. Kydd, 5
S. S. Lazarus, 4
K. S. Lee, 3
W. Lintz, 6R
L. Loewe, 3
T. P. Magill, 6
R. A. Moore, 4
E. Muntwyler, 2
J. O. Pinkston, 1, 3
L. Procita, 3
A. E. Sobel, 2
R. S. Speirs, 1
E. E. Suckling, 1
B. W. Volk, 4
M. Wachstein, 4
M. Walzer, 6
A. S. Wiener, 6
W. D. Wosilait, 2

BUFFALO
C. M. Ambrus, 1, 3
J. L. Ambrus, 1, 3
N. Back, 3
J. M. Benforado, 3
M. R. Blair, Jr., 3
E. M. Bridge, 3
C. Caruthers, 2
G. Clark, 1
F. R. Griffith, Jr., 1R, 5R
J. R. Hughes, 1
D. T. Karzon, 6
G. F. Koepf, 1
W. D. Langley, 2
C. U. Lowe, 4
E. Mihich, 3
J. F. Mohn, 6
E. Neter, 6
C. A. Nichol, 2
A. Nisonoff, 2
W. E. Noell, 1
D. Pressman, 2, 6
H. Rahn, 1
D. S. Riggs, 3
J. T. Roberts, 1
S. Rodbard, 1
N. R. Rose, 6
F. Rosen, 5
M. Schlamowitz, 2
S. Shulman, 2
K. L. Terplan, 4
E. Witebsky, 6
Y. Yagi, 6

CARMEL
A. A. Epstein, 2R

CHAPPAQUA
A. F. Coburn, 6

COOPERSTOWN
C. A. Ashley, 4
J. Bordley, III, 1
C. V. Z. Hawn, 4
T. Peters, Jr., 2

DELMAR
W. R. Thompson, 2

DOUGLASTON
F. E. Kendall, 6

ELMHURST
A. L. Lieberman, 1

FISHERS
F. S. Robscheit-Robbins, 1, 4

FLUSHING
L. G. Parmer, 3

FOREST HILLS
A. G. White, 1

FREEPORT
M. Brin, 2, 5

GENEVA
D. B. Hand, 2
Z. I. Kertesz, 2

GREENPORT
C. J. DeBoer, 6
G. C. Robinson, 1R

IRVINGTON-ON-HUDSON
H. F. Wood, 6

ISLIP
N. Rakieten, 1

ITHACA
J. A. Baker, 4
R. H. Barnes, 2, 5
C. L. Comar, 2, 5
L. J. Daniel, 2, 5
E. A. Delwiche, 2
R. W. Dougherty, 1
H. H. Dukes, 1
J. A. Dye, 1
M. Gibbs, 2
H. M. Hauck, 5
G. P. Hess, 2
F. W. Hill, 5
R. W. Holley, 2
F. A. Johnston, 5
H. S. Liddell, 1
J. K. Loosli, 5
L. A. Maynard, 2R, 5
C. M. McCay, 2, 5
W. L. Nelson, 2, 5
L. C. Norris, 2, 5
A. L. Romanoff, 1
H. A. Scheraga, 2
M. L. Scott, 2, 5
S. E. Smith, 5
R. H. Wasserman, 5
H. H. Williams, 2, 5
L. D. Wright, 2, 5
C. M. Young, 5

KEW GARDENS
J. E. Nadler, 3
A. Weil, 4

LARCHMONT
R. C. Hockett, 2

LONG ISLAND CITY
M. Weichsel, 6

MALBA
H. J. Spoor, 1

MALONE
C. P. Sherwin, 2R

MANHASSET
W. H. Sebrell, Jr., 2, 5

MASPETH
B. L. Oser, 5

MINEOLA
E. Ponder, 1

MOUNT KISCO
R. Cole, 4R, 6R

MOUNT VERNON
W. H. F. Addison, 1R
S. J. Piliero, 1

NEW CITY
W. D. Gray, 3

NEW HYDE PARK
J. Greenberg, 1

NEW ROCHELLE
H. E. Dubin, 2
W. A. Jacobs, 2R, 3R
C. Neumann, 1

NEW YORK CITY
L. L. Abell, 2
H. A. Abramson, 1, 2
F. L. Adler, 6
E. H. Ahrens, Jr., 2
F. M. Allen, 1R, 4
N. Alrazuler, 1
V. E. Amassian, 1
M. Anchel, 2
D. H. Andersen, 4
W. Antopol, 4
R. M. Archibald, 2
J. F. Artusio, Jr., 3
S. Baez, 1
M. E. Balis, 2
R. K. Barclay, 2
H. G. Barker, 1
H. L. Barnett, 1
R. C. Batterman, 3
W. S. Battersby, 1
J. H. Bauer, 4
E. F. Beach, 2, 5
E. L. Becker, 1
R. R. Becker, 2
J. A. Behre, 2
J. W. Bellville, 3
B. Benacerraf, 6
M. B. Bender, 1
A. Bendich, 2
J. G. Benton, 3
B. N. Berg, 4
E. Y. Berger, 1
A. W. Bernheimer, 6
M. Bevans, 4
M. Bier, 2
O. Bodansky, 2, 3
R. W. Bonsnes, 2
E. Borek, 2
L. J. Boyd, 3
S. E. Bradley, 1
H. L. Bradlow, 2
G. Brawerman, 2
F. Brink, Jr., 1
W. A. Briscoe, 1
D. W. Bronk, 1
V. B. Brooks, 1
G. B. Brown, 2
A. Brunschwig, 4
L. Buchbinder, 6R
N. M. Buckley, 1
J. C. Bugher, 4
H. H. Bunzell, 2R
J. H. Burchenal, 5
M. L. Caldwell, 2, 5
W. G. B. Casselman, 1
McK. Cattell, 1, 3
L. F. Cavalieri, 2
L. R. Cerecedo, 2, 5
H.-T. Chang, 1
L. F. Chapman, 1
E. Chargaff, 2
H. A. Charipper, 1
M. W. Chase, 6
H. Chasis, 1
L. R. Christensen, 6
J. G. Chusid, 1
L. J. Cizek, 1
D. A. Clarke, 3
R. D. Cole, 2
R. A. Cooke, 6
J. M. Cooperman, 5
A. F. Coumand, 1
D. Cowen, 4
L. C. Craig, 2
G. Dalldorf, 4, 6
B. S. Danes, 1
B. Davidow, 3
D. F. Davies, 1
C. R. Dawson, 2
R. C. de Bodo, 1, 3
P. F. de Gara, 6
A. C. DeGraff, 1, 3
C. G. de Gutierrez-Mahoney, 1, 4
I. J. Deyrup, 1
N. A. DiSalvo, 1
Z. Dische, 2
A. R. Dochez, 4R, 6R
V. P. Dole, 1
W. W. Douglas, 1, 3
E. F. Dubois, 1R, 3R, 5R
R. J. Dubos, 6
M. E. Dunn, 1
V. duVigneaud, 2, 5
M. Dworetzky, 6
D. D. Dziewiatkowski, 2
H. A. Eder, 2
D. J. Edwards, 1R
L. W. Eichna, 1
M. A. Eisenberg, 2
H. Elfman, 1
J. T. Ellis, 4
S. A. Ellison, 6
S. Englard, 2
B. F. Erlanger, 2
C. R. Falk, 6

NEW YORK

S. J. Farber, 1
P. Feigelson, 2
E. E. Fischel, 6
E. H. Fishberg, 2
A. P. Fishman, 1
P. J. Fodor, 2
N. C. Foot, 4R
C. L. Fox, Jr., 1R, 6
J. J. Fox, 2
C. E. French, 3
G. H. Fried, 4
C. Friend, 6
H. W. Fritts, Jr., 1
M. J. Frumin, 3
D. K. Fukushima, 2
C. Funk, 2R, 5
M. Galdston, 1
T. F. Gallagher, 2
A. L. Garbat, 6
H. S. Gasser, 1, 3
S. Gelfan, 1
A. Gellhorn, 3
H. Gershberg, 1
A. O. Gettler, 2R
A. Gilman, 1, 3
C. Gilvarg, 2
G. B. J. Glass, 1
W. F. Goebel, 2, 6
H. Gold, 3
A. Goldfeder, 1
A. J. Goldforb, 1
W. Goldring, 1
E. D. Goldsmith, 1
A. S. Gordon, 1
I. Graef, 4
S. Graff, 2
C. H. Graham, 1
S. Granick, 2
I. Greenwald, 2R, 5R
M. I. Gregersen, 1
J. D. Gregory, 2
R. L. Greif, 1
A. C. Groschke, 5
P. R. Gross, 1
J. Grossman, 1
H. Grundfest, 1
F. Gudematch, 1
F. R. Gurd, 2
A. B. Gutman, 2
C. Haig, 1
H. Haimovici, 1
S. P. Halbert, 6
L. D. Hamilton, 4
C. W. Hampel, 1
F. M. Hanger, 6
M. Hardy, 1
B. Harrow, 2R
H. K. Hartline, 1
J. E. Hawkins, Jr., 1
T. Hayashi, 1
O. M. Helff, 1
J. B. Herrmann, 3
S. G. Hershey, 3
J. G. Hilton, 1
J. C. Hinsey, 1
E. Hirschberg, 2
G. K. Hirst, 6
H. D. Hoberman, 2
E. S. Hodgson, 1
P. F. A. Hoefer, 1
F. G. Hofmann, 3
F. Hollander, 1, 2
L. E. Holt, Jr., 2, 5
F. L. Horsfall, Jr., 6
R. D. Hotchkiss, 2
R. W. Houde, 3
C. Howe, 6
J. M. Hundley, 5
J. Hurwitz, 2
M. S. Ingalls, 6
H. L. Jaffe, 4
J. W. Jailer, 1
H. D. Janowitz, 1
M. E. Jarvik, 3
M. A. Jessaitis, 2
N. Jolliffe, 1, 5
C. W. Jungeblut, 4, 6
E. A. Kabat, 2, 6
D. A. Kamofsky, 3
M. Karshan, 2
H. Kaunitz, 4
A. Kellner, 4
R. H. Kessler, 1
H. D. Kesten, 4
A. S. Keston, 2
A. C. Kibrick, 2
J. G. Kidd, 4, 6
E. D. Kilbourne, 6
C. G. King, 2, 5
B. Kisch, 1
S. J. Klebanoff, 2
I. S. Kleiner, 1, 2R, 3R, 5R
N. Kopeloff, 6R
L. Komgold, 6
H. P. Krieger, 1
H. D. Kruse, 2, 5
L. S. Kubie, 4
M. Kunitz, 2R
H. G. Kunkel, 2
S. Kupfer, 1
H. S. Kupperman, 3
M. Kuschner, 4
N. T. Kwit, 3
A. Lajtha, 2
R. C. Lancefield, 6
C. Landis, 1
K. Lange, 4
D. L. Larson, 6
H. D. Lauson, 1
H. S. Lawrence, 6
R. E. Lee, 1
R. A. Lehman, 3
D. Lehr, 3
E. S. L'Esperance, 6
S. Z. Levine, 5
M. F. Levitt, 1
M. Levitz, 2
M. Levy, 2
R. L. Levy, 3
S. Lieberman, 2
P. A. Lief, 3
A. Linker, 2
M. Lipkin, 1
F. Lipmann, 2
D. P. C. Lloyd, 1
R. O. Loebel, 1R
O. Loewi, 3H
M. E. Lombardo, 2
I. M. London, 2
R. Lorente de Nó, 1
W. E. Loring, 4
M. H. Loveless, 6
B. W. Low, 2
F. V. Lucas, 4
W. K. Maas, 2
J. MacLeod, 1
L. I. Malis, 1
I. Mandl, 2
L. C. Mark, 3
P. A. Marks, 2
C. D. May, 5
A. Mazur, 2
M. McCarty, 6
P. D. McMaster, 4, 6
R. C. Mellors, 2, 4, 6
D. B. Melville, 2
M. Mendlowitz, 1
R. B. Merrifield, 2
F. A. Mettler, 1
K. Meyer, 2
A. T. Milhorat, 1, 3, 5
J. G. Millichap, 3
A. E. Mirsky, 2
W. Modell, 3
A. E. Moore, 6
D. H. Moore, 1
S. Moore, 2
C. Morgan, 6
R. S. Morison, 1
M. C. Morris, 6
E. H. Mosbach, 2
I. M. Mountain, 6
G. E. Murphy, 4
D. Nachmansohn, 1, 2
W. L. Nastuk, 1
S. Natelson, 2
J. M. Neill, 6
C. T. Nelson, 6
J. B. Nelson, 4
J. M. Nelson, 2R
N. Nelson, 2
W. O. Nelson, 1
I. Neuwirth, 2, 3
S.-H. Ngai, 3
H. C. Nicholson, 1
F. F. Nord, 2
A. B. Novikoff, 2
S. Ochoa, 2
P. K. Olitsky, 6R
E. L. Opie, 1R, 4R, 6R
E. T. Oppenheimer, 1
L. R. Orkin, 3
L. G. Ortega, 4
M. I. Osterhout, 1
W. J. V. Osterhout, 1
G. T. Pack, 1
E. D. Palmes, 1
E. M. Papper, 3
I. A. Parfentjev, 6
P. Y. Paterson, 6
J. M. Pearce, 4
O. H. Pearson, 2
P. B. Pearson, 2, 5
G. E. Perlmann, 2
M. L. Petermann, 2
F. S. Philips, 3
E. P. Pick, 3
R. F. Pitts, 1
H. Pollack, 1, 5
H. Popper, 4
D. P. Purpura, 1
J. R. Rachele, 2
E. Racker, 2
C. Ragan, 1
E. P. Ralli, 1, 5
M. M. Rapport, 2
S. Ratner, 2
R. W. Rawson, 1
W. Redisch, 1
J. Reilly, 3
E. H. Reisner, Jr., 5
C. Ressler, 2
P. Reznikoff, 1
C. P. Rhoads, 4
D. W. Richards, Jr., 1
G. W. Richter, 4
M. N. Richter, 4
W. F. Riker, Jr., 3
S. H. Rinzler, 3
D. Ritzenberg, 2
T. M. Rivers, 4, 6
J. Roberts, 3
R. O. Roblin, Jr., 2
M. Rockstein, 1
L. Roizin, 4
R. W. Root, 1
W. S. Root, 1
H. M. Rose, 6
R. S. Rosenfeld, 2
P. Rous, 4
E. A. Rovenstine, 3
A. Sandow, 1
W. H. Sawyer, 1, 3
B. Schick, 6
J. Schiller, 1
H. A. Schneider, 5
S. S. Schneierson, 6
M. Schubert, 3
W. J. Schubert, 2
M. Schweizer, 1
D. P. Seecof, 4
B. C. Seegal, 4, 6
D. Seegal, 6
S. Seifter, 2
C. B. Shaffer, 3
D. Sheehan, 1
D. Shemin, 2
L. B. Shettles, 1
R. E. Shope, 4
G. Shwartzman, 4, 6
P. Siekevitz, 2
E. J. Simon, 2
I. Singer, 4
H. P. Smith, 1, 4
H. W. Smith, 1, 2
L. W. Smith, 4
M. H. D. Smith, 6
S. C. Smith, 5
K. C. Smithburn, 6
S. E. Snyderman, 5
H. H. Sobotka, 2, 5
M. Sonenberg, 2
T. H. Spaet, 1
W. M. Sperry, 2
D. B. Sprinson, 2
E. E. Sproul, 4
J. M. Steele, 1
W. H. Stein, 2
C. A. Stetson, 4, 6
F. W. Stewart, 4
W. B. Stewart, 4
C. C. Stock, 2
G. C. H. Stone, 2
H. J. Strecker, 2
J. Y. Sugg, 6
R. C. Swan, Jr., 1
J. V. Taggart, 1
H. H. Tallan, 2
I. Tamm, 6
S. W. Tanenbaum, 2
E. L. Tatum, 2
C. M. Taylor, 5
R. M. Taylor, 1
A. W. Thomas, 2
L. Thomas, 6
D. D. Thompson, 1
W. S. Tillett, 6
H. W. Toolan, 4
W. Trager, 4
F. B. Traub, 6
J. Travell, 3
W. Troll, 2
J. W. Uhr, 6
L. J. Unger, 6
H. B. van Dyke, 1, 3
T. B. Van Itallie, 5
F. S. Vogel, 4
P. Vogel, 6
W. Waelsch, 2
I. H. Wagman, 1
G. E. Wakerlin, 1, 3
W. W. Walcott, 1
S. C. Wang, 1, 3
R. C. Warner, 2
B. Webster, 5
R. Weggia, 1, 3
A. J. Weil, 6
V. L. Weimar, 1
B. Weiss, 2
P. A. Weiss, 1
B. Weissmann, 2
J. T. Weld, 6
E. A. Werber, 6
G. Werner, 3
L. G. Wesson, Jr., 1
G. W. Wheeler, 6R
A. White, 2, 5
S. L. Wilens, 4

NEW YORK – NORTH CAROLINA

T. L. Willmon, 1
V. J. Wilson, 1
J. Wittenberg, 2
A. Wolf, 4
H. G. Wolff, 1, 3
D. W. Woolley, 2, 3, 5
S. B. Wortis, 1
T.-F. Yu, 1, 3
S. Zamenhof, 2
H. L. Zauder, 3
H. M. Zimmerman, 4
M. B. Zucker, 1
B. W. Zweifach, 1

APO, NEW YORK CITY
R. B. Lindberg, 6

NORWICH
R. J. Main, 1
M. F. Paul, 2
W. T. Rockhold, 3

OAKDALE
L. K. Riggs, 2R

OSSINING
L. C. Miller, 3

OWEGO
L. W. Famulener, 6R

PEARL RIVER
P. H. Bell, 2
H. R. Cox, 6
R. W. Cunningham, 3
I. S. Danielson, 2
E. C. DeRenzo, 2
R. H. Hall, 2
W. Hausmann, 2
B. L. Hutchings, 2
T. H. Jukes, 2, 5
J. T. Litchfield, Jr., 3
E. L. Patterson, 2
R. R. Roepke, 3
G. R. Sharpless, 5
E. L. R. Stokstad, 2, 5
W. L. Williams, 2

PORT WASHINGTON
N. Molomut, 6
W. H. Pearlman, 2

POUGHKEEPSIE
R. E. Conklin, 1

QUEENS VILLAGE
F. Co Tui, 3

RENSSELAER
A. Arnold, 5
E. W. Dennis, 3, 6
L. Grumbach, 1
J. O. Hoppe, 3
A. M. Lands, 1, 3
J. R. Lewis, 3
F. P. Luduena, 3
E. W. McChesney, 1
M. L. Tainter, 1, 3
R. L. Thompson, 6

RICHMOND HILL
H. Blumberg, 3, 5

ROCHESTER
E. F. Adolph, 1
E. L. Alling, 4
K. I. Altman, 2
S. R. Ames, 2
W. F. Bale, 1
H. A. Blair, 1
W. R. Bloor, 1R, 2R
C. G. Bly, 4
E. S. Boyd, 3
G. W. Casarett, 4
E. A. K. Culler, 1R
V. F. Di Stefano, 3
A. L. Dounce, 2
N. D. Embree, 2
D. W. Fassett, 3
W. O. Fenn, 1
E. L. Gasteiger, Jr., 1
R. Gerschman, 1
E. LeB. Gray, 1
P. L. Harris, 2, 5
F. L. Haven, 2
W. B. Hawkins, 4
E. E. Hawley, 5
E. E. Hays, 2
J. R. Hinshaw, 4
H. C. Hodge, 2, 3
J. W. Howland, 1, 4
J. P. Lambooy, 2
G. V. Marinetti, 2
K. E. Mason, 5
E. A. Maynard, 3
W. S. McCann, 2R, 5
L. L. Miller, 2
H. R. Morgan, 6
M. Morrison, 2
P. E. Morrow, 3
J. J. Morton, 4R
J. Murlin, 1R, 2R, 5R
E. S. Nasset, 1, 5
W. F. Neuman, 2, 3
T. R. Noonan, 1
J. L. Orbison, 4
H. E. Pearse, 4
J. C. Peskin, 1
W. T. Pommerenke, 1
H. L. Rosenthal, 2
A. Rothstein, 1, 3
K. Salomon, 2
J. U. Schlegel, 1
J. K. Scott, 4
W. K. Smith, 1
J. N. Stannard, 1, 3
E. H. Stotz, 2
R. Terry, 4
J. H. Vaughan, 6
G. H. Whipple, 1R, 4R

ROCKVILLE CENTER
E. J. Quinn, 2R, 5R

ROME
A. La Belle, 1

ROSLYN
S. A. Berson, 1

ROSLYN HEIGHTS
A. L. Florman, 6
E. Perlman, 6

SARATOGA SPRINGS
J. M. Reiner, 1

SCARSDALE
E. Baumann, 2R
R. J. Block, 2, 5
T. E. Boyd, 1
C. N. Frey, 5

SOUTHAMPTON
W. W. Brandes, 4

SUNMOUNT
F. Klemperer, 2

SYRACUSE
P. B. Armstrong, 1
W. R. Boss, 1
H. L. Dickison, 3
H. F. Diermeier, 3
M. S. Dooley, 3R
A. E. Farah, 3
H. A. Feldman, 6
J. A. Halsted, 4
J. B. Hoekstra, 3
B. V. Jager, 6
F. P. Knowlton, 1R
V. F. Lindeman, 1
C. W. Lloyd, 1
S. Mallov, 3
J. M. McKibbin, 2
G. K. Moe, 1
O. H. Müller, 1
J. B. Preston, 3
C. N. Remy, 2
B. R. Rennick, 3
D. A. Richert, 2
J. S. Robb, 1, 3
W. R. Ruegamer, 2
O. W. Sartorius, 1
M. P. Schulman, 2
H. M. Tepperman, 1
J. Tepperman, 1, 3
I. C. Wells, 2
W. W. Westerfeld, 2
P. N. Witt, 3
V. J. Wulff, 1

TARRYTOWN
B. F. Daubert, 2

TROY
G. M. Sickles, 6

TUCKAHOE
E. J. de Beer, 3
C. H. Ellis, 1
I. Goodman, 2
G. H. Hitchings, 2
R. Koster, 3
S. Norton, 3
J. C. Seed, 3

UPTON
V. P. Bond, 1
G. C. Cozzias, 4
E. P. Cronkite, 1
H. J. Curtis, 1
L. K. Dahl, 4
R. Drew, 6
L. E. Farr, 4
L. V. Hankes, 2
C. H. W. Hirs, 2
W. L. Hughes, 2
J. R. M. Innes, 4
J. R. Klein, 2
D. E. Koshland, Jr., 2
M. E. Koshland, 6
P. G. Lefevre, 1
S. W. Lippincott, 4
M. Maxfield, 1
L. F. Nims, 1
E. A. Popenoe, 2
J. S. Robertson, 1
I. L. Schwartz, 1
W. W. Shreeve, 2
R. Steele, 2
R. D. Stoner, 4
D. D. Van Slyke, 2

UTICA
N. S. Olsen, 1, 2

VALHALLA
M. H. Williams, Jr., 1

VALLEY STREAM
D. R. Axelrod, 1
L. C. Chesley, 1

WHITE PLAINS
D. A. Boroff, 6

YONKERS
R. H. Broh-Kahn, 1
G. Unger, 1

NORTH CAROLINA

BILTMORE
M. H. Givens, 1R, 2R

CHAPEL HILL
C. E. Anderson, 2
B. Baggett, 2
K. M. Brinkhous, 4
T. C. Butler, 3
W. J. Cromartie, 4
T. Z. Csaky, 1, 3
E. C. Curnen, 6
F. W. Ellis, 3
J. H. Ferguson, 1
C. W. Gottschalk, 1
J. B. Graham, 4
E. M. Irvin, 2
J. L. Irvin, 2
R. D. Langdell, 4
G. P. Manire, 6
W. S. McClellan, 1
A. T. Miller, Jr., 1
G. D. Peaick, 4
J. H. Perlmutt, 1
J. H. Schwab, 6
H. Tauber, 2
W. J. Waddell, 3
L. G. Welt, 1

CHARLOTTE
F. G. Germuth, 4, 6

DURHAM
R. D. Baker, 4
J. W. Beard, 4, 6
R. F. Becker, 1
F. Bernheim, 2, 3
W. E. De Turk, 3
G. S. Eadie, 1, 3
F. L. Engel, 1
J. W. Everett, 1
McC. Goodall, 1
K. S. Grimson, 1, 3
F. G. Hall, 1
P. Handler, 2, 5
J. P. Hendrix, 3
W. E. Hull, 1
H. Kamin, 2
W. Kempner, 1
H. Lowenbach, 1
G. Margolis, 4
J. E. Markee, 1
F. D. McCrea, 3
J. R. Overman, 6
K. E. Penrod, 1
B. Schmidt-Nielsen, 1
K. Schmidt-Nielsen, 1
G. W. Schwert, 2
A. G. Smith, 4
D. T. Smith, 5, 6
E. A. Stead, Jr., 1
H. M. Taylor, 4
J. V. Warren, 1
K. M. Wilbur, 1
J. W. Wilson, 1

FLATROCK
H. Laurens, 1R

GREENSBORO
C. I. Reed, 1R
M. F. Williams, 1

HENDERSONVILLE
R. E. Remington, 2R, 5R

NORTH CAROLINA – NORTH DAKOTA – OHIO – OKLAHOMA – OREGON – PENNSYLVANIA

MOUNT PLEASANT
A. E. Woodward, 1R

RALEIGH
H. W. Garren, 1
C. H. Hill, 5
G. Matrone, 5
G. H. Satterfield, 2, 5
S. T. Walton, 6
G. H. Wise, 5

SALISBURY
J. E. Gregory, 6

WINSTON-SALEM
C. Arton, 2
J. R. R. Bobb, 1
A. B. Denison, Jr., 1
H. D. Green, 1, 3
R. A. Groat, 1
J. M. Little, 1, 3
M. P. Spencer, 1
M. A. Swanson, 2
W. A. Wolff, 2

NORTH DAKOTA

FARGO
D. F. Eveleth, 2

GRAND FORKS
W. E. Cornatzer, 2
B. De Boer, 1, 3
H. E. Ederstrom, 1

OHIO

AKRON
L. Catron, 4

ATHENS
F. A. Hellebrandt, 1R

CHAGRIN FALLS
R. W. Eckstein, 1

CINCINNATI
G. H. Acheson, 3
H. Braunstein, 4
H. Burlington, 1
J. J. Cohen, 1
G. M. Conzelman, Jr., 3
M. L. Cooper, 6
D. I. Crandall, 2
G. Eckstein, 1
M. H. Fischer, 1, 2R
E. A. Gall, 4
G. M. Guest, 2
L. G. Herrmann, 4
F. F. Heyroth, 2R
D. E. Jackson, 1R, 3R
J. B. Kahn, Jr., 3
R. A. Kehoe, 1
B. H. Landing, 4
M. A. Logan, 2
W. D. Lotspeich, 1
M. B. Macht, 1
F. H. Mattson, 2, 5
B. F. Miller, 2
Z. B. Miller, 2
C. A. Mills, 1, 2
F. J. Murray, 6
L. J. Pecora, 1
I. Ruchman, 6
A. B. Sabin, 6
L. H. Schmidt, 2, 3
J. Scholler, 3
C. C. Smith, 3
H. E. Stokinger, 3, 6
J. L. Svirbely, 3
S. Tashiro, 1R, 2R
E. F. Van Maanen, 3
R. L. Vick, 3
C. K. Weichert, 1
H. W. Werner, 3
C. D. West, 1
B. C. Wexler, 4
R. J. Young, 5

CIRCLEVILLE
A. W. Bosworth, 2R, 5R

CLEVELAND
E. R. Arquilla, 6
C. S. Beck, 4
R. M. Berne, 1
E. H. Bloch, 6
J. W. Blunt, 4
P. E. Boyle, 4
J. J. Clemmons, 4
C. Cooper, 2
A. C. Corcoran, 1, 3
F. W. Denny, Jr.. 6
J. H. Dingle, 6
R. Dominguez, 1
E. E. Ecker, 4, 6
J. B. Garst, 2
H. Gest, 2
H. S. Ginsberg, 6
H. Goldblatt, 1, 4
D. A. Goldthwait, 2
E. Haas, 2
D. B. Hackel, 1, 4
H. K. Hellerstein, 1
H. Hirschmann, 2
H. A. Hunsicher, 5
M. H. Kaplan, 6
N. Kaufman, 4
T. D. Kinney, 4
J. Kleinerman, 4
S. Kolecsky, 4
L. O. Krampitz, 2, 6
J. Lamer, 2
P. S. Lavik, 2
M. Leon, 6
J. R. Leonards, 2
I. H. Lepow, 6
C. Leuchtenberger, 4
S. Levey, 2, 5
M. N. Levy, 1
L. A. Lewis, 1
G. M. C. Masson, 1
F. R. Mautz, 1
J. W. McCubbin, 1
R. D. Moore, 4
A. R. Moritz, 4
I. H. Page, 1, 2
R. F. Parker, 4, 6
C. H. Rammelkamp, 6
O. A. Ross, 4
I. Rothchild, 1
H. Rudney, 2
H. Z. Sable, 2
H. Sachs, 2
W. Sakami, 2
P. R. Salerno, 3
G. Sayers, 1, 3
L. Share, 1
R. A. Shipley, 1
E. A. Smith, 1
T. Sollmann, 1R, 2R, 3
J. Spizizen, 2, 6
A. B. Stavitsky, 6
J. R. Stern, 2
E. W. Sutherland, Jr., 2, 3
H. E. Swim, 2
M. F. Utter, 2
J. T. Wearn, 1
R. J. P. Wedgwood, 6
J. M. Werle, 1
C. J. Wiggers, 1R, 3R
H. G. Wood, 2
G. W. Wright, 1

CLEVELAND HEIGHTS
W. J. Kolff, 1
F. C. Robbins, 6
N. C. Wetzel, 5

COLUMBUS
C. A. Angerer, 1
J. M. B. Bloodworth, Jr., 4
E. Bozler, 1
J. B. Brown, 2, 5
K. A. Brownell, 1
E. T. Carter, 1
G. M. Curtis, 4R
F. E. Deatherage, 2
C. A. Doan, 4
M. Dodd, 6
R. C. Grubbs, 1
F. A. Hartman, 1R
F. A. Hitchcock, 1, 5
M. Koehne, 5R
C. D. Leake, 1, 3
M. A. Lessler, 1
W. G. Myers, 1
E. Ogden, 1
M. B. Patton, 5
L. A. Sapirstein, 1
S. Saslaw, 6
D. G. Scarpelli, 4
G. Y. Shinowara, 2
R. W. Stacy, 1
T. S. Sutton, 5
J. F. Tomashefski, 1
J. E. Varner, 2
E. von Haam, 4
G. C. Webster, 2
B. K. Wiseman, 4
M. L. Wolfrom, 2

DAYTON
J. F. Hall, Jr., 1
J. W. Heim, 1
R. F. Kline, 1
H. O. Parrack, 1

EUCLID
G. L. Farrell, 1

NEW CARLISLE
E. P. Hiatt, 1

OXFORD
W. C. McNelly, 1

TOLEDO
E. L. Burns, 4
B. Steinberg, 4, 6

WOOSTER
O. G. Bentley, 5
J. W. Hibbs, 5
W. E. Krauss, 2, 5
A. L. Moxon, 2

YELLOW SPRINGS
M. D. Armstrong, 2
P. W. K. Rothemund, 2
P. Webb, 1

YOUNGSTOWN
A. Goudsmit, 1

OKLAHOMA

NORMAN
S. H. Wender, 2

OKLAHOMA CITY
R. M. Bird, 1
R. Caputto, 2
M. R. Everett, 2
R. H. Furman, 1
J. A. Hagans, 3
A. A. Hellbaum, 1, 3
M. N. Huffman, 2
W. E. Jaques, 4
W. Joel, 4
A. Kurtz, 2
R. W. Payne, 3
J. A. Schilling, 4
F. Sheppard, 2
H. A. Shoemaker, 3
P. W. Smith, 1, 3
A. N. Taylor, 1
R. E. Trucco, 2
S. Wolf, 1

STILLWATER
W. D. Gallup, 2
V. G. Heller, 2R, 5R
L. M. Henderson, 2, 5
R. MacVicar, 2, 5

OREGON

CORVALLIS
J. S. Butts, 2, 5
V. H. Cheldelin, 2, 5
M. L. Fincke, 5
J. R. Haag, 2, 5
H. M. Krueger, 1, 3
R. W. Newburgh, 2
C. A. Storvick, 5
C. H. Wang, 2
R. Wulzen, 1R

EUGENE
F. J. Reithel, 2
B. T. Scheer, 1
A. L. Soderwall, 1

OSWEGO
A. C. Kirchhof, 3

PORTLAND
G. M. Austin, 1
C. H. Beatty, 1
W. D. Blake, 1
J. M. Brookhart, 1
N. A. David, 3
R. S. Dow, 1
H. F. Haney, 1
I. A. Manville, 1
H. S. Mason, 2
E. L. McCawley, 3
E. L. Oginsky, 2
R. D. Peterson, 2
N. M. Phatak, 3
F. B. Queen, 4
B. B. Ross, 1
L. E. Thomas, 2
W. R. Todd, 2, 5
A. R. Tunturi, 1
J. T. Van Bruggen, 2
E. S. West, 2

PENNSYLVANIA

BALA-CYNWYD
W. J. Rashkind, 1

BRADFORD
A. Dury, 1

PENNSYLVANIA

BRISTOL
C. V. Smythe, 2

BRYN MAWR
E. A. Bliss, 6
A. N. Richards, 1R, 2R, 3R

CHELTENHAM
H. Goldberg, 1

GETTYSBURG
J. H. Hewetson, 5

GLENSIDE
D. B. McN. Scott, 2

HAVERFORD
R. E. Forster, II, 1

HAVERTOWN
R. A. McLean, 3

JOHNSVILLE
J. D. Hardy, 1
R. W. Lawton, 1
A. M. Stoll, 1

MEDIA
L. J. Saul, 1

NEWTOWN SQUARE
M. G. Sevag, 2, 6

NORRISTOWN
J. F. McClendon, 1R, 2R, 5R

PHILADELPHIA
P. M. Apfelman, 6
J. D. Aronson, 4, 6
D. M. Aviado, Jr., 1, 3
C. Bachman, 2
D. H. Baeder, 3
W. W. Baker, 3
T. C. Barnes, 1, 3
W. J. Beckfield, 4
F. B. Benjamin, 1
M. A. Bennett, 2
W. S. Blakemore, 1
A. Bondi, 6
S. Y. Botelho, 1
P. A. Bott, 1, 2
M. J. Boyd, 2
C. Breedis, 4
H. Brieger, 3
B. A. Briody, 6
J. R. Brobeck, 1
F. P. Brooks, 1
C. P. Brown, 6
B. Calesnick, 3
A. Cantarow, 1, 3
W. A. Cassel, 6
W. W. Chambers, 1
B. Chance, 1, 2
F. C. Charalampous, 2
H. F. Chase, 3
P. O. Chatfield, 1
E. R. Clark, 1R
J. K. Clark, 1
M. F. Clarke, 5
S. S. Cohen, 2
D. R. Coman, 4
H. L. Coon, Jr., 1
L. Cook, 3
J. M. Coon, 3
A. P. Crosley, Jr., 3
S. A. D'Angelo, 1
R. E. Davies, 2
R. H. De Meio, 2
J. Dempsher, 3
V. G. Dethier, 1
D. K. Derweiler, 1
J. R. Di Palma, 1, 3
F. C. Dohan, 1
D. L. Drabkin, 2, 5
R. D. Dripps, 1, 3
I. N. Dubin, 4
A. B. Du Bois, 1
J. E. Eckenhoff, 1
M. R. Ehrenstein, 2
W. Ehrich, 4
H. J. Eichel, 2
G. Eisenman, 1
J. R. Elkinton, 1
S. Ellis, 3
J. D. Evans, 1
G. E. Farrar, Jr., 3
M. Fay, 2
E. J. Fellows, 3
L. K. Ferguson, 4
L. B. Flexner, 1, 2
J. A. Flick, 6
N. F. Floyd, 2
R. Freter, 6
M. H. F. Friedman, 1
W. L. Gaby, 6
P. George, 2
T. S. Githens, 1R
J. M. Glassman, 3
D. R. Goddard, 2
J. W. Goddard, 4
K. Goodner, 6
W. G. Gordon, 2
H. Green, 2
S. M. Greenberg, 5
E. M. Greisheimer, 1
S. Gurin, 2
P. György, 2, 5
J. H. Hafkenschiel, Jr., 1, 3
H. R. Hafkenbring, 1
C. Harris, 4
T. N. Harris, 6
J. A. Harrison, 6
N. Haugaard, 2, 3
F. X. Hausberger, 1
L. V. Heilbrunn, 1
A. E. Heming, 3
W. Henle, 6
J. S. Hepburn, 2R
A. Y. Hillier, 1
D. E. Holtkamp, 3
S. M. Horvath, 1
O. Horwitz, 1
J. Hughes, 1
K. Hummeler, 6
M. H. Jacobs, 1
J. H. Jones, 2, 5
A. Kandel, 3
A. S. Kaplan, 6
F. Karush, 2, 6
L. A. Kazal, 2
J. J. Kocsis, 3
G. B. Koelle, 3
J. A. Kolmer, 6R
S. A. Komarov, 1
H. Koprowski, 6
C. P. Krantz, 3
E. B. Krumbhaar, 1R, 4R
C. J. Lambertsen, 1, 3
T. F. Lavine, 2
D. H. Lewis, 1
W. H. Lewis, 1R
G. N. Ling, 1
I. Live, 6
E. Lodholz, 1R
S. Lorber, 1
F. D. Lukens, 1
M. B. Lurie, 4, 6
C. M. MacLeod, 6
R. W. Manthei, 3
J. Marsh, 2
G. J. Martin, 2
P. A. Mattis, 3
S. McD. McCann, 1
G. P. McCouch, 1
F. H. McCutcheon, 1
M. McCutcheon, 4
W. F. McLimans, 6
T. L. McMeekin, 2
G. Medes, 2
V. Menkin, 1, 4, 6
R. E. Miller, 6
L. C. Mills, Jr., 3
H. Montgomery, 1
J. H. Moyer, III, 3
S. Mudd, 1, 4, 6
P. Oesper, 2
M. J. Oesterling, 2
E. A. Ohler, 1
M. J. Oppenheimer, 1
W. M. Parkins, 1
K. E. Paschkis, 1
E. K. Patterson, 2
S. M. Peacock, Jr., 1
R. Penneys, 1
L. H. Peterson, 1
I. J. Pincus, 1
H. L. Price, 1, 3
H. L. Ratcliffe, 4
I. S. Ravdin, 1, 4
E. A. Reed, 1
S. P. Reimann, 1, 4
J. G. Reinhold, 2
M. Reynolds, 1
J. E. Rhoads, 1
F. Riedels, 3
C. Riegel, 2
H. W. Robinson, 2
K. Rodahl, 1
C. S. Rose, 2
H. Rosen, 3
O. Rosenthal, 2
J. L. d'A. Roth, 1
J. S. Roth, 2
D. O. Rudin, 1
R. J. Rutman, 2
C. F. Schmidt, 1, 3
J. J. Schneider, 2
J. Schultz, 2
D. Scott, Jr., 1
J. C. Scott, 1
F. B. Seibert, 2
J. Seifter, 3
R. W. Sevy, 1
H. Shapiro, 1
H. Shay, 1
W. B. Shelley, 1
H. A. Sloviter, 2
J. C. Small, 4
L. Smith, 2
J. Smolens, 6
S. Sorof, 2
E. H. Spaulding, 6
J. N. Spencer, 3
E. A. Spiegel, 1
M. Spiegel-Adolf, 2
M. A. Spirtes, 3
J. J. Spitzer, 1
J. M. Sprague, 1
G. F. Springer, 6
R. D. Squires, 1
W. C. Stadie, 2R
E. Staple, 2
I. Starr, 1, 3
J. A. Stekol, 2, 5
M. Strassman, 2
F. M. Sturtevant, 3
F. W. Sunderman, 2
D. H. Tedeschi, 3
W. H. Telfer, 6
P.-O. Therman, 1
R. Tislow, 1, 3
L. M. Tocantins, 1
G. Toennies, 2
J. C. Touchstone, 2
R. G. Tucker, 5
L. Tuft, 6
H. M. Vars, 2, 5
J. M. Waldron, 1
L. Weil, 2
S. Weinhouse, 2
C. Weiss, 4, 6
H. A. Wendel, 3
M. P. Wiedeman, 1
F. J. Wiercinski, 1
D. W. Wilson, 1R, 2
M. G. Wohl, 5
I. T. Zeckwer, 1R, 4R
I. Zeidman, 4
F. Zilliken, 2
C. A. Zittle, 2

PITTSBURGH
R. Abrams, 2
A. E. Axelrod, 2, 5, 6
L. V. Beck, 1, 3
H. S. Belding, 1
R. Bentley, 2
H. Bloch, 6
F. S. Cheever, 6
L. A. Cohen, 1
G. J. Cox, 2, 5
L. H. Cretcher, 2R
L. H. Criep, 6
T. S. Danowski, 1
N. C. Davis, 2
F. J. Dixon, Jr., 4, 6
A. Edelmann, 1
E. Ellenbogen, 2
R. S. Farr, 4, 6
E. J. Fedor, 1
J. D. Feldman, 4
M. A. Fischer, 2
B. Fisher, 1
E. R. Fisher, 4
F. F. Foldes, 3
M. Fuld-Conderman, 2
O. Gawron, 2
H. M. Gezon, 6
M. I. Gluckman, 3
R. C. Grauer, 4
P. Gross, 4
W. McD. Hammon, 4, 6
E. R. Hart, 3
K. Hofmann, 2
T. K. Kruse, 1, 3R
W. J. Kuhns, 6
G. R. Lacy, 4R
M. A. Lauffer, Jr., 2
J. Leighton, 4
J. H. Lewis, 1
I. Lieberman, 2
A. S. Marrazzi, 1, 3
S. P. Masouredis, 1, 6
P. H. Maurer, 6
R. H. McCoy, 2, 5
W. McElroy, 2
P. L. McLain, 1, 3
I. R. McManus, 2
I. A. Mirsky, 1
T. J. Moran, 4
C. Moses, 1
J. D. Myers, 1
H. Noll, 2
R. E. Olson, 2, 5
H. H. Permar, 4
A. G. Renfrew, 2
C. H. W. Ruhe, 1
J. E. Salk, 6
N. H. Sloane, 2
A. H. Stock, 6
F. A. Taylor, 2
F. I. Tsuji, 2
J. J. Vazquez, 4
W. O. Weigle, 6
J. S. Youngner, 6

RADNOR
F. W. Bernhart, 5

PUERTO RICO – RHODE ISLAND – SOUTH CAROLINA – SOUTH DAKOTA – TENNESSEE – TEXAS

R. M. Tomarelli, 2, 5

STATE COLLEGE
R. A. Dutcher, 2R, 5R
E. B. Forbes, 1R, 5R
E. D. Wilson, 5

SWARTHMORE
S. Kletzien, 5

TELFORD
E. S. Meadelson, 1

UNIVERSITY PARK
A. A. Benson, 2
A. Black, 5
R. V. Boucher, 5
J. W. Bratzler, 5
M. L. Dobbs, 5
K. H. Fisher, 5
N. B. Guerrant, 2, 5
M. F. Mallette, 2
R. C. Miller, 5
R. L. Pike, 5
R. W. Swift, 5

VILLANOVA
E. L. Foltz, 1, 3

WARREN
Z. Stary, 2

WAYNE
W. P. Boger, 3
A. E. Livingston, 1R, 3R

WEST POINT
K. H. Beyer, Jr., 1, 3
J. Charney, 2
E. W. J. DeMaar, 3
R. A. Harte, 5
M. R. Hilleman, 6
J. W. Huff, 2
O. C. Liu, 6
H. M. Peck, 3
N. P. Plotnikoff, 3
C. C. Porter, 2
R. B. Stebbins, 4
K. M. Stevens, 6
C. A. Stone, 3
A. A. Tytell, 2, 6
V. G. Vernier, 3
C. A. Winter, 1, 3

WILLOW GROVE
C. F. Gell, 1

YEADON
W. C. Foster, 1

PUERTO RICO

HATO REY
R. M. Reinecke, 1

RIO PIEDRAS
L. J. Roberts, 5

SAN JUAN
C. F. Asenjo, 2, 5
C. B. Casas, 1
M. Goettsch, 2, 5
J. Szepsenwol, 1
D. B. Tyler, 1, 3

RHODE ISLAND

JAMESTOWN
S. L. Wright, 2

PROVIDENCE
P. F. Fenton, 1, 5
H. Kabat, 1
E. H. Leduc, 4
C. Pfaffmann, 1
L. A. Riggs, 1
F. G. Sherman, 1
C. A. Stuart, 6
J. W. Wilson, 1, 4

SOUTH CAROLINA

CHARLESTON
T. G. Bernthal, 1
T. B. Calhoon, 1
T. D. Darby, 3
F. W. Kinard, 1
M. H. Knisely, 1
J. A. Richardson, 3
R. P. Walton, 3
E. F. Woods, 3

CLEMSON
E. J. Lease, 5

COLUMBIA
J. D. Reese, 4

NEWBERRY
F. N. Martin, Jr., 3

SOUTH DAKOTA

BROOKINGS
O. E. Olson, 2
C. Schuck, 5

VERMILLION
M. A. Heinrich, Jr., 3
F. E. Kelsey, 3
F. K. O. Kelsey, 3
D. M. MacCanon, 1
W. O. Read, 1

TENNESSEE

CHATTANOOGA
S. H. Auerbach, 4

COOKEVILLE
J. T. Skinner, 2

KNOXVILLE
G. T. Barry, 2
F. L. MacLeod, 2, 5
G. MacLeod, 2R, 5R
S. Rogers, 4
S. R. Tipton, 1

MEMPHIS
R. H. Alden, 1
G. Barlow, 1
D. A. Brody, 1
S. R. Bruesch, 1
N. R. Di Luzio, 1
A. D. Dulaney, 6
W. B. Dunham, 6
C. C. Erickson, 4
A. Ganz, 3
O. S. Gibbs, 3
W. M. Hale, 4, 6
M. Klein, 4
R. E. Koeppe, 2
K. A. Kuiken, 2
N. D. Lee, 2
H. S. Loucks, 1
G. D. Lumb, 4
E. L. McCandless, 1
D. B. Morrison, 2
T. P. Nash, Jr., 2
R. R. Overman, 1
Van B. Philpot, Jr., 4
J. P. Quigley, 1, 3
H. C. Schmeisser, 4
C. W. Sheppard, 1
D. H. Spruit, 4, 6
G. B. Spurr, 1
L. Van Middlesworth, 1
W. L. Williams, 4
J. L. Wood, 2
R. A. Woodbury, 1, 3
J. M. Young, 4
D. B. Zilversmit, 1

MOUNTAIN HOME
W. Fleischmann, 1

NASHVILLE
H. V. Aposhian, 3
A. D. Bass, 3
F. R. Blood, 1
E. B. Bridgforth, 5
M. T. Bush, 3
R. J. Cazort, 3
J. G. Coniglio, 2
L. W. Cunningham, 2
W. J. Darby, 2, 5
H. Goldie, 6
F. Gollan, 1
C. M. Greer, 3
P. F. Hahn, 1, 4
E. G. High, 5
W. C. Holland, 3
L. Hurwitz, 3
P. D. Lamson, 1R, 3R
G. V. Mann, 5
W. J. McGanity, 5
G. R. Meneely, 1, 4
H. C. Meng, 1
A. S. Minot, 1
V. A. Najjar, 2
E. V. Newman, 1
C. R. Park, 1, 2
J. H. Park, 2
J. T. Park, 2
W. N. Pearson, 5
R. L. Post, 1
B. H. Robbins, 3
C. S. Robinson, 2R
D. T. Rolfe, 1
J. L. Shapiro, 4
O. Touster, 2
H. D. West, 2
C. W. Woodruff, 5

OAK RIDGE
N. G. Anderson, 1
S. F. Carson, 2
W. E. Cohn, 2
C. C. Congdon, 4
D. G. Doherty, 2
M. I. Dolin, 2
N. Gengozian, 6
A. Hollaender, 1
G. C. Kyker, 2
T. Makinodan, 6
G. D. Novelli, 2
T. T. Odell, Jr., 1
C. H. Steffee, 4
A. C. Upton, 4
E. Volkin, 2

TEXAS

ABILENE
F. W. Dunn, 2

AUSTIN
J. E. Davis, 1, 3
R. E. Eskin, 2
J. W. Foster, 2
J. M. Ravel, 2
L. J. Reed, 2
A. F. Riggs, 2
W. Shive, 2
C. G. Skinner, 2
H. M. Sweeney, 1
R. J. Williams, 2
O. Wyss, 2

COLLEGE STATION
J. R. Couch, 2, 5
H. O. Kunkel, 2
C. M. Lyman, 2, 5
R. Reiser, 2
L. R. Richardson, 5

DALLAS
J. G. Bishop, 1
W. P. Blackmore, 3
W. W. Burr, Jr., 2
R. T. Capps, 3
A. Goth, 3
A. Grollman, 1, 3
S. Haberman, 6
F. Harrison, 1
R. W. Lackey, 1
M. F. Mason, 2
E. E. Muirhead, 1, 4
J. C. Porter, 1
A. F. Reid, 1
F. T. Rogers, 1R
M. B. Slomka, 3
R. T. Smith, 4, 6
S. E. Sulkin, 6
H. C. Tidwell, 2, 5
J. C. Vanatta, 1

DENTON
F. I. Scoular, 5

FORT WORTH
H. H. Beard, 2

GALVESTON
C. R. Allen, 1
D. B. Calvin, 1, 2
G. A. Emerson, 3
R. L. Gregory, 1
M. M. Guest, 1
C. E. Hall, 1
A. E. Hansen, 4, 5
G. R. Herrmann, 4R
H. C. Hopps, 4, 6
J. B. Nash, 3
W. W. Nowinski, 1
A. A. Ormsby, 2
A. Packchanian, 6
M. Pollard, 4
C. M. Pomerat, 1
E. L. Porter, 1
E. J. Poth, 1
R. H. Rigdon, 4
A. Ruskin, 1
G. R. Seaman, 1
H. G. Swann, 1
H. F. Wiese, 5

HOUSTON
E. C. Alvord, Jr., 4
J. Awapara, 2
E. Beerstecher, Jr., 2
K. L. Burdon, 6
V. M. Doctor, 2
R. Edelberg, 1
J. R. Elliott, 1
C. Fortier, 1
J. A. Greene, 1
T. H. Greiner, 3

TEXAS – UTAH – VERMONT – VIRGINIA – WASHINGTON – WEST VIRGINIA – WISCONSIN

A. C. Griffin, 2
R. Guillemin, 1
B. Halpert, 4
C. A. Handley, 3
G. M. Higgins, 5R
H. E. Hoff, 1
R. A. Huggins, 1, 3
R. B. Hurlbert, 2
D. Karp, 3
A. S. Keats, 3
P. Kellaway, 1
A. Kirschbaum, 4
S. Kit, 2
B. M. Levy, 4
J. L. Melnick, 6
J. L. Rabinowitz, 2
W. O. Russell, 4
R. A. Seibert, 3
E. L. Smith, 1
S. L. Steelman, 2
R. V. Talmage, 1
J. B. Walker, 2

HUNT
A. E. Meyer, 1R

RANDOLPH AFB
R. W. Bancroft, 1
O. O. Benson, Jr., 1
H. G. Clamann, 1
R. T. Clark, Jr., 1
J. D. Fulton, 6
H. B. Gerstner, 1
L. J. Milch, 3
H. Strughold, 1
P. C. Tang, 1
S. Wood, Jr., 4

SAN ANTONIO
H. F. Adler, 1
E. A. Blair, 1
H. J. Schattenberg, 4
R. D. Templeton, 1
N. T. Werthessen, 1
H. S. Wigodsky, 1

TEMPLE
N. C. Hightower, Jr., 1
E. J. Mason, 4

UTAH

LOGAN
C. Biddulph, 1
D. A. Greenwood, 3
L. E. Harris, 5

OGDEN
W. A. Bennett, 4
S. P. Hayes, 6

PROVO
D. M. Donaldson, 6
L. P. Vernon, 2

SALT LAKE CITY
H. L. Borison, 1, 3
W. H. Carnes, 4
G. E. Cartwright, 5
S. R. Dickman, 2
T. F. Dougherty, 1
C. Edwards, 1
K. B. Eik-Nes, 1
D. W. Esplin, 3
C. Fenning, 1
E. G. Fingl, 3
L. P. Gebhardt, 6
L. S. Goodman, 1, 3
F. D. Gunn, 4
R. B. Harvey, 1
S. C. Harvey, 3
H. Hecht, 1
R. L. Hill, 2
C. C. Hunt, 1
L. W. Jarcho, 1
R. S. Jones, 4
J. R. Kimmel, 2
W. S. Loewe, 3
S. Marcus, 6
A. R. Martin, 1
C. McNeil, 6
E. R. Perl, 1
G. W. E. Plaut, 2
L. T. Samuels, 2, 3, 5
E. L. Smith, 2
J. D. Spikes, 1
M. L. Sweat, 2
E. A. Swinyard, 3
J. L. Van Lancker, 4
H. R. Warner, 1
N. Weissman, 2
W. G. Wiest, 2
M. M. Wintrobe, 4
D. M. Woodbury, 3
L. A. Woodbury, 1

VERMONT

BRADFORD
S. B. Hooker, 6R

BURLINGTON
A. H. Chambers, 1
R. W. Coon, 4
L. V. Crowley, 4
E. C. Gjessing, 2
C. Hanna, 3
R. Korson, 4
E. Lepeschkin, 1
W. H. Macmillan, 3
H. B. Pierce, 2, 5
W. Raab, 1
W. Van B. Robertson, 2
A. H. Schein, 2
F. J. M. Sichel, 1
D. J. Smith, 1, 3
W. L. Wilson, 1

WEST BRATTLEBORO
H. A. Schroeder, 1

VIRGINIA

ARLINGTON
G. K. Cotts, 3
C. W. Shilling, 1
F. Sperling, 3

BLACKSBURG
R. W. Engel, 5
R. F. Miller, 5

CHARLOTTESVILLE
W. P. Anslow, Jr., 1, 2
E. D. Brand, 3
S. W. Britton, 1R
J. R. Cash, 4
A. Chanutin, 2
E. L. Corey, 1
A. E. Feller, 6
C. L. Gemmill, 1, 2, 3
D. R. H. Gourley, 1, 3
C. Hoch-Ligeti, 4
V. P. Hollander, 2
W. S. Jordan, 6
D. W. Kupke, 2
S. Ludewig, 2
R. T. Major, 2
R. W. McGilvery, 2
Q. N. Myrvik, 6
G. C. Pitts, 1

FALLS CHURCH
W. E. Chamberlain, 1
I. Cornman, 1
M. A. Fink, 6
L. W. Hazleton, 3

HERNDON
G. Woodard, 3

NORFOLK
E. H. Lanphier, 1

PEDLAR MILLS
E. R. Long, 4R

RICHMOND
L. D. F. Abbott, Jr., 2
F. L. Apperly, 4
R. S. Cunningham, 1R
L. E. Edwards, 1
R. H. Egdahl, 4
J. K. Finnegan, 3
E. Fischer, 1
J. C. Forbes, 2
H. B. Haag, 3
N. M. Hajek-Nichols, 1
H. Hoch, 2
E. C. Hoff, 1
E. G. Huf, 1
D. M. Hume, 4
E. L. Jackson, 3
P. S. Larson, 1, 3
H. McKennis, Jr., 2, 3
L. A. Mounter, 2
J. K. Palmer, 2
J. L. Patterson, Jr., 1
R. W. Ramsey, 1
R. B. Smith, Jr., 3
J. H. Weatherby, 3

STANARDSVILLE
H. Silvette, 1, 3

WASHINGTON

FORT STEILACOOM
M. A. Kennard, 1
F. W. Maire, 1

PULLMAN
L. L. Campbell, Jr., 2
D. S. Farner, 1
J. R. Gorham, 4, 6
L. L. Madsen, 5
J. McGinnis, 5
C. M. Stevens, 2

SEATTLE
E. P. Benditt, 4
H. S. Bennett, 1
A. Bodansky, 2R
E. A. Boyden, 1
K. Buettner, 1
L. D. Carlson, 1
E. D. Clark, 2R
J. M. Dille, 3
C. A. Evans, 6
N. B. Everett, 1
G. Falk, 1, 3
D. D. Feller, 1
C. A. Finch, 1
E. H. Fischer, 2
B. W. Gabrio, 2
P. D. Goldsworthy, 2
D. J. Hanahan, 2
J. P. Hannon, 1
H. N. Harkins, 1
C. G. Heller, 1
A. Horita, 3
F. M. Huennekens, 2
E. G. Krebs, 2
T. A. Loomis, 3
B. Mackler, 2
D. F. Magee, 1, 3
H. C. Mason, 6
J. C. Mathies, 2
L. E. Morris, 3
H. Neurath, 2, 6
H. D. Patton, 1
R. T. Prehn, 4
J. I. Rowntree, 5
T. C. Ruch, 1
R. F. Rushmer, 1
A. M. Scher, 1
A. L. Towe, 1
B. M. Wagner, 4
C. P. Wangeman, 3
A. A. Ward, Jr., 1
R. S. Weiser, 6
T. C. West, 3
H. Wheelon, 1
H. R. Whiteley, 2
P. E. Wilcox, 2
R. H. Williams, 4
J. W. Woodbury, 1
A. C. Young, 1

TACOMA
L. R. Goldbaum, 3

UNDERWOOD
J. E. Halver, 5

WEST VIRGINIA

MARTINSBURG
L. Swell, 2

MORGANTOWN
A. C. Higginbotham, 1
R. F. Krause, 2
G. R. McKinney, 3
D. W. Northup, 1
H. Patrick, 5
J. C. Stickney, 1
E. J. Van Liere, 1
D. T. Watts, 3

WISCONSIN

DE FOREST
R. C. Herrin, 1

MADISON
K. Akert, 1
R. A. Alberty, 2
L. Anderson, 2
D. M. Angevine, 4
R. L. Baldwin, 2
C. A. Baumann, 2, 5
H. Beinert, 2
D. T. Berman, 6
H. R. Bird, 5
R. M. Bock, 2
H. D. Bouman, 1
R. H. Burris, 2
P. F. Clark, 6R
P. P. Cohen, 2
F. L. Crane, 2
C. W. Crampton, 3
H. F. Deutsch, 2
C. A. Elvehjem, 2, 5
J. D. Ferry, 2
D. S. Goldman, 2

WISCONSIN – WYOMING – CANADA – MEXICO

D. E. Green, 2
C. J. Gubler, 5
A. E. Harper, 2, 5
C. Heidelberger, 2
L. E. Hokin, 2
M. R. Irwin, 6
M. J. Johnson, 2
P. Kaesberg, 2
F. L. Kozelka, 3
S. A. Kuby, 2
J. J. Lalich, 4
H. A. Lardy, 2
E. M. Larsen, 1
G. A. Le Page, 2
K. P. Link, 2
B. J. Luyet, 1
G. J. Mannering, 3
W. H. McShan, 2
W. J. Meek, 1R
R. K. Meyer, 1
E. C. Miller, 2
J. A. Miller, 2
J. W. Miller, 3
P. R. Morrison, 1, 2
G. C. Mueller, 2
Q. R. Murphy, 1
C. Olson, Jr., 4
O. S. Orth, 1, 3
R. E. Parks, Jr., 3
H. T. Parsons, 2R, 5R
W. H. Peterson, 2R, 5R
P. H. Phillips, 2, 5
J. W. Porter, 2
Van R. Potter, 2
J. M. Price, 2
H. Reich, 2
M. S. Reynolds, 5
H. P. Rusch, 4
H. J. Sallach, 2
C. V. Seastone, Jr., 6
F. E. Shideman, 3
A. A. Siebens, 1
F. Skoog, 2
M. A. Stahmann, 2
H. Steenbock, 2R, 5
W. E. Stone, 1
F. M. Strong, 2
M. L. Sunde, 5
L. J. Teply, 2, 5
H. A. Waisman, 2
S. J. Wakil, 2
D. L. Walker, 6
G. H. Wang, 1
J. L. Way, 3
J. N. Williams, Jr., 2
J. W. Williams, 2
P. W. Wilson, 2
H. R. Wolfe, 6
C. N. Woolsey, 1
W. B. Youmans, 1, 3

MARSHFIELD
R. D. Taylor, 1

MILWAUKEE
C. H. Altshuler, 4
H. Beckman, 3
J. C. Bock, 2R
J. Y. Chen, 3
L. S. Cunningham, 2
J. H. Fodden, 4
G. W. Gray, 3
B. Kassell, 2
H. M. Klitgaard, 1
M. Laskowski, 2
H. Levine, 2, 5
S. A. Morell, 2
A. J. Quick, 2, 3
P. M. Roll, 2
B. H. Schlomovitz, 1
J. J. Smith, 1
W. D. Winters, 3

WOOD
L. H. Hamilton, 1

WYOMING

LARAMIE
I. Rosenfeld, 2

CANADA

ALBERTA

EDMONTON
H. B. Collier, 2
A. W. Downs, 1R
L. W. McElroy, 5
J. Tube, 2

RALSTON
M. K. McPhail, 3

BRITISH COLUMBIA

VANCOUVER
E. C. Black, 1
P. Constantinides, 1
D. H. Copp, 1
E. E. Daniel, 3
C. E. Dolman, 6
J. G. Foulks, 1, 3
S. M. Friedman, 1
H. G. Khorana, 2
R. A. MacLeod, 2
W. J. Polglase, 2
H. V. Rice, 1
H. L. A. Tarr, 2
H. E. Taylor, 4

MANITOBA

WINNIPEG
G. E. Delory, 2R
J. Doupe, 1
P. E. Dresel, 3
J. A. Hildes, 1
M. Nickerson, 1, 3
F. D. White, 2R

NEW BRUNSWICK

LANCASTER
E. A. G. Branch, 4

ST. JOHN
R. J. Collins, 3

NOVA SCOTIA

HALIFAX
W. W. Hawkins, 2
J. G. Kaplan, 1
E. R. M. Kay, 2
C. B. Weld, 1
E. G. Young, 2

LUNENBURG
A. B. Macallum, 2R

ONTARIO

CHALK RIVER
G. C. Butler, 2

GUELPH
H. D. Branion, 5

KINGSTON
J. M. R. Beveridge, 5
E. M. Boyd, 3
G. H. Ettinger, 1
R. H. More, 4
M. E. M. Sawyer, 1
R. E. Semple, 1

LEAMINGTON
M. L. Menten, 1R, 4

LONDON
A. C. Burton, 1
K. K. Carroll, 2
J. B. Collip, 1, 2
C. W. Gowdey, 3
F. C. Heagy, 2
G. W. Manning, 1
E. G. D. Murray, 6
R. L. Noble, 1
J. C. Paterson, 4
R. J. Rossiter, 2
G. W. Scavnicky, 1, 3
J. A. F. Stevenson, 1
K. P. Strickland, 2
R. A. Waud, 3

ORANGEVILLE
V. H. K. Moochouse, 1R

OTTAWA
J. R. Beaton, 2
J. A. Campbell, 5
A. Desmarais, 1
L. P. Dugal, 1
J. Gibbard, 6
L. Greenberg, 6
J. S. Hart, 1
R. M. Hochster, 2
S. F. Kitchen, 6
F. C. Lu, 3
B. B. Migicovsky, 2
J. F. Morgan, 2
C. A. Morrell, 3
K. T. Murray, 5
L. B. Pett, 5
L. I. Pugsley, 2

PORT HOPE
C. C. Benson, 2R

TORONTO
E. Baer, 2
C. H. Best, 1, 2
R. B. Bromiley, 1
M. H. Brown, 6
J. Campbell, 1
W. R. Campbell, 2R
S. L. Cohen, 2
H. Cullumbine, 3
T. G. H. Drake, 5
J. K. W. Ferguson, 1, 3
A. M. Fisher, 2
K. C. Fisher, 1
E. S. Goranson, 1
R. E. Haist, 1
A. W. Ham, 4
J. D. Hamilton, 4
C. S. Hanes, 2
A. Hunter, 2R
J. Hunter, 1
W. H. Johnson, 1
W. Kalow, 3
C. C. Lucas, 2
G. H. W. Lucas, 2, 3
J. F. Manery, 1
J. Markowitz, 1
W. D. McFarlane, 2
E. W. McHenry, 2, 5
B. A. McLaren, 5
F. R. Miller, 1R
F. C. Monkhouse, 1
E. C. Robertson, 5
D. A. Scott, 2R
E. A. Sellers, 1
C. O. Siebenmann, 3
F. A. Sunahara, 1
N. B. Taylor, 1
H. Wasteneys, 2R
A. M. Wynne, 2

QUEBEC

HULL
C. E. Rice, 6

MACDONALD COLLEGE
E. W. Crampton, 5
L. E. Lloyd, 5

MONTREAL
I. T. Beck, 3
B. G. Benfey, 3
W. Bourne, 3
J. S. L. Browne, 1
A. S. V. Burgen, 1, 3
B. D. Burns, 1
R. V. Christie, 1
R. A. Cleghorn, 1
O. F. Denstedt, 2
S. Dworkin, 1
K. A. C. Elliott, 2
V. Fredette, 6
H. H. Jasper, 1
H. Lubinski, 6
F. C. MacIntosh, 1, 3
J. D. McColl, 3
K. I. Melville, 3
E. Page, 1
J. M. Parker, 3
W. G. Penfield, 1
J. H. Quastel, 2
H. Selye, 1
T. L. Sourkes, 2
D. L. Thomson, 2
E. H. Venning, 2
D. R. Wood, 3

QUEBEC CITY
G. A. Bergeron, 1
R. Bernard, 1
D. Fairbairn, 2
J. A. LeBlanc, 1

SASKATCHEWAN

SASKATOON
R. W. Begg, 2
L. B. Jaques, 1, 3

MEXICO

MEXICO CITY D. F.
E. C. del Pozo, 1
F. Guerra, 3
J. J. Izquierdo, 1
C. Mendez, 3
R. Mendez, 3
E. G. Pardo, 3
A. Rosenblueth, 1
A. Zaffaroni, 2

MONTERREY
J. Pisanty O., 1

SAN LUIS POTOSI
R. Villarreal, 1

OUTSIDE NORTH AMERICA

OUTSIDE NORTH AMERICA

ARGENTINA

V. Deulofeu, 2
B. Houssay, 1H
L. F. Leloir, 2

AUSTRALIA

A. Bolliger, 2
J. C. Eccles, 1H
J. F. A. Sprent, 6

BELGIUM

F. Bremer, 1H
J. P. Henry, 1
C. Heymans, 3

BRAZIL

O. Bier, 6
J. G. Lacorte, 6
M. Rocha e Silva, 3
J. R. Valle, 3

BRITISH WEST INDIES

I. F. S. Mackay, 1

CHILE

M. Alcamirano, 1

CHINA

H. C. Hou, 5
C.-Y. Sung, 3

COLOMBIA

F. R. Hunter, 1
K. Mezey, 3

DENMARK

E. Aaes-Jorgensen, 5
H. Dam, 2, 5
E. Jacobsen, 3

ENGLAND

E. D. Adrian, 1H
M. L. Anson, 2
E. L. Beckman, 1
J. H. Burn, 3
A. L. Copley, 1
H. H. Dale, 3H
E. de Savitsch, 4
O. G. Edholm, 1
M. P. Fitzgerald, 1R
J. H. Gaddum, 3
A. V. Hill, 1H
R. Hodes, 1
A. Hodgkin, 1H
T. E. King, 2
V. Massey, 2
F. J. W. Roughton, 1H
H. Sinclair, 5

FINLAND

P. K. Roine, 5

FRANCE

A. Bussard, 6
R. L. Cahen, 3
H. E. Foster, 6R
P. Grabar, 6
J. Oudin, 6
M. Raynaud, 6
R. L. Zwemer, 1

GERMANY

O. H. Gauer, 1
B. Hoelscher, 1
S. Rapoport, 2
R. Thauer, 1
K. von Frisch, 1H
A. Wollenberger, 3

GREECE

J. Caminopetros, 6
A. S. Dontas, 3
G. Joachimoglu, 3

GUATEMALA

G. Arroyave, 5
R. Bressani, 5
N. S. Scrimshaw, 1, 5

HOLLAND

B. Mendel, 2

HONG KONG

C. C. Gruhzit, 3
L. G. Kilborn, 1

INDIA

H. N. Chatterjee, 6
R. N. Chopra, 3H
N. N. De, 3
E. D. Mason, 1
B. Mukerji, 3
P. S. Sarma, 5

INDONESIA

A. Knudson, 2R, 5

IRAN

H. A. Reimann, 4

IRELAND

J. W. Harman, 4

ISRAEL

D. Elson, 2
M. C. Shelesnyak, 1
T. Winnick, 2

ITALY

M. Ajazzi-Mancini, 3

JAPAN

O. Hayaishi, 2
K. Kamijo, 3
Y. Kobayashi, 3
T. Ogata, 6

LEBANON

S. E. Kerr, 2

NORWAY

J. Molland, 3

PARAGUAY

T. P. Hughes, 6

PERU

C. Monge, 1H
V. Zapata-Ortiz, 3

PHILIPPINES

E. Bulatao, 1R
F. O. Santos, 5

POLAND

J. Kaulbersz, 1

SWEDEN

G. Liljestrand, 1H
S. Thesleff, 3

SWITZERLAND

W. R. Hess, 1H
H. Langemann, 3
R. Meier, 3
J. M. Posternak, 1
E. Rothlin, 3
A. Stoll, 3
T. Wagner-Jauregg, 2
W. Wilbrandt, 3

UNION OF SOUTH AFRICA

N. B. Dreyer, 3

URUGUAY

J. R. Totter, 2

U.S.S.R.

L. A. Orbeli, 1H

VIETNAM

G. J. Stein, 6

ALPHABETICAL LIST OF MEMBERS

American Physiological Society

ACTIVE MEMBERS

L. G. Abood
D. I. Abramson
H. A. Abramson
W. Adams
H. W. Ades
W. R. Adey
H. F. Adler
E. F. Adolph
E. D. Adrian (Hon.)
C. M. Agress
R. P. Ahlquist
C. Ajmone-Marsan
R. P. Akers
K. Akert
A. Albert
R. H. Alden
N. Alexander
R. S. Alexander
B. V. Alfredson
C. R. Allen
J. G. Allen
L. Allen
S. C. Allen
T. H. Allen
W. M. Allen
G. A. Alles
E. L. Alpen
M. Altamirano
P. D. Altland
N. Altszuler
A. S. Alving
V. E. Amassian
W. R. Amberson
C. M. Ambrus
J. L. Ambrus
E. M. Anderson
N. G. Anderson
R. S. Anderson
R. Andres
E. C. Andrus
C. A. Angerer
J. H. Annegers
W. P. Anslow, Jr.
M. H. Aprison
J. W. Archdeacon
P. B. Armstrong
W. D. Armstrong
E. B. Astwood
G. M. Austin
D. M. Aviado, Jr.
D. R. Axelrod
H. Bacchus
L. M. N. Bach
W. H. Bachrach
A. M. Baetjer
S. Baez
R. C. Bahn
P. Bailey
B. L. Baker
E. J. Baldes
W. F. Bale
R. W. Bancroft
P. Bard
A. C. Barger
H. G. Barker
S. B. Barker
G. Barlow
B. O. Barnes
T. C. Barnes
H. L. Barnett
D. H. Barron
R. G. Bartlett, Jr.
S. H. Bartley
D. E. Bass
J. B. Bateman
W. S. Battersby
W. Bauer
J. W. Bean
C. H. Beatty
L. V. Beck
E. L. Becker
R. F. Becker
E. L. Beckman
H. K. Beecher
A. R. Behnke
L. M. Beidler
H. S. Belding
M. B. Bender
F. B. Benjamin
A. L. Bennett
H. S. Bennett
L. L. Bennett
O. O. Benson, Jr.
T. H. Benzinger
J. R. Bergen
E. Y. Berger
G. A. Bergeron
N. I. Berlin
R. W. Berliner
R. Bernard
R. M. Berne
T. G. Bernthal
G. H. Berryman
S. A. Berson
C. H. Best
K. H. Beyer, Jr.
R. G. Bickford
C. Biddulph
R. J. Bing
R. M. Bird
J. H. Birnie
J. E. Birren
G. H. Bishop
J. G. Bishop
E. C. Black
E. A. Blair
H. A. Blair
J. R. Blair
W. D. Blake
W. S. Blakemore
B. B. Blivaiss
F. R. Blood
J. J. Blum
J. Blumenstock
J. R. R. Bobb
E. J. Boell
E. G. Boettiger
D. F. Bohr
V. C. Bohr
V. P. Bond
W. M. Booker
B. R. Boone
J. Bordley, III
H. L. Borison
W. R. Boss
S. Y. Botelho
P. A. Bott
D. J. Botts
H. D. Bouman
J. E. Bourque
H. Bourquin
W. J. Bowen
L. L. Boyarsky
T. E. Boyd
E. A. Boyden
E. Bozler
J. T. Bradbury
S. E. Bradley
W. B. Bradley
C. R. Brassfield
R. W. Brauer
E. Braunwald
M. A. B. Brazier
G. A. Brecher
F. Bremer (Hon.)
N. R. Brewer
F. Brink, Jr.
W. A. Briscoe
J. R. Brobeck
W. A. Brodsky
A. T. Brody
D. A. Brody
R. H. Broh-Kahn
R. B. Bromiley
D. W. Bronk
J. M. Brookhart
C. Brooks
C. McC. Brooks
F. P. Brooks
M. M. Brooks
V. B. Brooks
L. Brouha
D. E. S. Brown
E. Brown
E. B. Brown, Jr.
F. A. Brown, Jr.
G. W. Brown
J. H. U. Brown
R. V. Brown
J. S. L. Browne
K. A. Brownell
J. Brozek
A. M. Brues
S. R. Bruesch
J. M. Bruhn
H. D. Bruner
W. R. Bryan
W. C. Buchbinder
N. L. R. Bucher
N. M. Buckley
P. C. Bucy
K. Buettner
T. H. Bullock
C. A. Bunde
H. B. Burchell
H. O. Burdick
A. S. V. Burgen
H. Burlington
B. D. Burns
A. C. Burton
E. R. Buskirk
S. O. Byers
T. B. Calhoon
D. B. Calvin
B. Campbell
J. Campbell
A. Cantarow
A. Canzanelli
L. D. Carlson
E. B. Carmichael
L. Carmichael
F. G. Carpenter
E. T. Carter
C. B. Casas
W. G. B. Casselman
E. S. Castle
H. R. Catchpole
McK. Cattell
H. M. Cavert
L. E. Chadwick
I. L. Chaikoff
W. E. Chamberlain
A. H. Chambers
E. L. Chambers
L. A. Chambers
W. H. Chambers
W. W. Chambers
B. Chance
H.-T. Chang
M. C. Chang
J. L. Chapin
L. F. Chapman
H. A. Charipper
A. M. Chase
H. Chasis
P. O. Chatfield
K. K. Chen
L. C. Chesley
H. I. Chinn
K. L. Chow
R. V. Christie
L. Churney
J. G. Chusid
L. J. Cizek
H. G. Clamann
M. H. Clare
G. Clark
J. K. Clark
L. C. Clark, Jr.
R. T. Clark, Jr.
W. G. Clark
R. W. Clarke
R. A. Cleghorn
C. D. Clemente
C. F. Code
H. Cohen
J. J. Cohen
L. A. Cohen
C. Cohn
K. S. Cole
L. J. Cole
C. R. Collier
W. D. Collings
J. B. Collip
J. H. Comroe, Jr.
R. E. Conklin
H. L. Conn, Jr.
P. Constantinides
S. F. Cook
D. E. Copeland
A. L. Copley
D. H. Copp
A. C. Corcoran
E. L. Corey
I. Cornman
S. A. Corson
A. F. Cournand
B. G. Covino
F. N. Craig
L. A. Crandall, Jr.
P. F. Cranefield, Jr.
M. F. Crawford
F. Crescitelli
G. R. Crisler
J. M. Crismon
P. J. Crittenden
E. P. Cronkite
T. Z. Csaky
T. K. Cureton, Jr.
H. J. Curtis
E. Da Costa
R. G. Daggs
H. E. Dale
F. E. D'Amour

M. C. D'Amour
B. S. Danes
S. A. D'Angelo
T. S. Danowski
R. C. Darling
C. W. Darrow
H. W. Davenport
V. D. Davenport
D. F. Davies
P. W. Davies
A. K. Davis
H. Davis
J. E. Davis
J. O. Davis
R. C. de Bodo
B. De Boer
A. C. DeGraff
C. G. de Gutierrez-Mahoney
J. M. R. Delgado
F. del Greco
E. C. del Pozo
E. W. Dempsey
A. B. Denison, Jr.
J. S. Denslow
A. J. Derbyshire
A. Desmarais
V. G. Dethier
D. K. Detweiler
L. Dexter
F. L. Dey
I. Deyrup
D. B. Dill
N. R. Di Luzio
J. R. Di Palma
N. A. Di Salvo
E. L. Dobson
E. L. Dohan
V. P. Dole
R. Dominguez
L. B. Dotti
R. W. Doty
R. W. Dougherty
T. F. Dougherty
W. W. Douglas
J. Doupe
P. Dow
R. S. Dow
R. M. Dowben
W. L. Doyle
C. A. Dragstedt
L. D. Dragstedt
V. A. Drill
R. D. Dripps
R. L. Driver
D. R. Drury
A. B. Du Bois
L. P. Dugal
H. H. Dukes
M. E. Dunn
F. T. Dun
A. L. Dunn
R. P. Durbin
A. Dury
W. R. Duryee
S. Dworkin
J. A. Dye
A. J. Dziemian
G. S. Eadie
E. Eagle
D. P. Earle, Jr.
D. M. Easton
J. C. Eccles (Hon.)
J. E. Eckenhoff
G. Eckstein
R. W. Eckstein
R. Edelberg
I. S. Edelman
A. Edelmann
H. E. Ederstrom
O. G. Edholm
C. Edwards
J. G. Edwards
L. E. Edwards
L. W. Eichna
K. B. Eik-Nes
G. Eisenman
E. Eldred
H. Elftman
J. R. Elkinton
J. R. Elliott
C. H. Ellis
F. Elmadjian
L. H. Elwell
J. D. Emerson
F. L. Engel
C. Entenman
D. L. Espe
H. E. Essex
C. F. Essig
G. H. Ettinger
F. G. Evans
G. T. Evans
J. D. Evans
T. C. Evans
E. V. Evarts
J. W. Everett
N. B. Everett
W. J. Eversole
J. L. Fahey
G. Falk
S. J. Farber
D. S. Farner
G. L. Farrell
J. I. Farrell
E. J. Fedor
G. A. Feigen
D. D. Feller
J. M. Felts
W. O. Fenn
C. Fenning
P. F. Fenton
F. P. Ferguson
J. Ferguson
J. H. Ferguson
J. K. W. Ferguson
B. G. Ferris, Jr.
E. S. Fetcher
D. Fetter
J. Field, II
G. F. Filley
C. A. Finch
J. C. Finerty
W. M. Firor
E. Fischer
M. H. Fischer
B. Fisher
K. C. Fisher
A. P. Fishman
W. Fleischmann
L. B. Flexner
P. P. Foa
G. E. Folk, Jr.
E. L. Foltz
A. Forbes
W. H. Forbes
F. M. Forster
R. E. Forster, II
R. P. Forster
C. Fortier
W. C. Foster
J. G. Foulks
W. S. Fowler
C. L. Fox, Jr.
K. Frank
F. E. Franke
R. M. Fraps
A. S. Freedberg
L. W. Freeman
N. E. Freeman
S. Freeman
M. J. Fregly
W. H. Freygang, Jr.
H. B. Friedgood
M. Friedman
M. H. Friedman
M. H. F. Friedman
S. M. Friedman
H. W. Fritts, Jr.
I. B. Fritz
W. J. Fry
F. A. Fuhrman
J. L. Fuller
G P. Fulton
J. F. Fulton
M. G. F. Fuortes
R. H. Furman
A. P. Gagge
R. Galambos
M. Galdston
W. F. Ganong
W. H. Gantt
E. D. Gardner
H. W. Garren
H. S. Gasser
F. X. Gassmer
E. L. Gasteiger, Jr.
M. Gaudino
O. H. Gauer
R. Gaunt
S. Gelfan
C. F. Gell
E. Gellhorn
C. L. Gemmill
R. W. Gerard
E. B. Gerbeim
R. Gerschman
H. Gershberg
J. W. Gersten
H. B. Gerstner
G. O. Gey
F. A. Gibbs
A. Gilman
A. S. Gilson, Jr.
G. B. J. Glass
A. J. Glazko
N. Glickman
A. D. Glinos
F. R. Goetzl
H. Goldberg
H. Goldblatt
A. Goldfeder
A. J. Goldforb
A. Goldin
D. E. Goldman
S. Goldring
W. Goldring
E. D. Goldsmith
M. S. Goldstein
F. Gollan
W. T. Goodale
McC. Goodall
H. C. Goodman
L. S. Goodman
E. S. Goranson
Albert S. Gordon
Archer S. Gordon
D. B. Gordon
R. E. Gosselin
C. W. Gottschalk
A. Goudsmit, Jr.
D. R. H. Gourley
A. L. Grafflin
C. H. Graham
H. T. Graham
F. Grande
E. R. Grant
R. Grant
W. C. Grant
E. Le B. Gray
J. S. Gray
S. W. Gray
A. Graybiel
P. O. Greeley
D. M. Green
H. D. Green
J. D. Green
J. Greenberg
R. Greenberg
C. H. Greene
J. A. Greene
H. Greengard
R. O. Greep
M. I. Gregersen
D. E. Gregg
R. L. Gregory
R. L. Greif
E. M. Greisheimer
R. G. Grenell
D. R. Griffin
K. S. Grimson
J. H. Grindlay
R. A. Groat
D. Grob
F. S. Grodins
A. Grollman
E. G. Gross
Jack Gross
Jerome Gross
P. Gross
J. Grossman
M. I. Grossman
R. C. Grubbs
L. Grumbach
H. Grundfest
F. Gudernatsch
M. M. Guest
R. Guillemin
V. Guillemin, Jr.
R. M. Guttman
A. C. Guyton
D. B. Hackel
F. J. Haddy
Z. Hadidian
J. H. Hafkenschiel, Jr.
H. R. Hafkenbring
P. F. Hahn
C. Haig
H. Haimovici
R. E. Haist
N. M. Hajek-Nichols
F. Halberg
J. Haldi
C. E. Hall
F. G. Hall
J. F. Hall, Jr.
V. E. Hall
G. A. Hallenbeck
W. C. Halstead
C. E. Hamilton
J. B. Hamilton
L. H. Hamilton
W. F. Hamilton
C. W. Hampel
J. K. Hampton, Jr.
S. W. Handford
H. F. Haney
J. P. Hanson
E. Hardenbergh
James D. Hardy
James Daniel Hardy
M. Hardy
K. Hare
H. N. Harkins
P. M. Harmon
A. S. Harris
S. C. Harris
F. Harrison
H. C. Harrison
J. S. Hart
W. M. Hart
H. K. Hartline
V. B. Hartman
A. McG. Harvey
R. B. Harvey
C. C. Hassett
A. B. Hastings
F. X. Hausberger
P. B. Hawk
J. E. Hawkins, Jr.
E. W. Hawthorne
E. C. Hay
T. Hayashi
J. M. Hayman, Jr.
F. W. Haynes
C. Haywood
J. Z. Hearon

H. Hecht
O. M. Hechter
A. H. Hegnauer
L. V. Heilbrunn
J. W. Heim
P. Heinbecker
O. M. Helff
A. A. Hellbaum
C. G. Heller
J. H. Heller
H. K. Hellerstein
A. Hemingway
C. D. Hendley
E. D. Hendley
E. Henneman
J. P. Henry
A. F. Henschel
C. M. Herget
T. Hernandez
J. F. Herrick
R. C. Herrin
L. P. Herrington
R. Hertz
A. B. Hertzman
W. R. Hess (Hon.)
A. W. Hetherington
E. A. Hewitt
E. P. Hiatt
J. B. Hickam
W. A. Hiestand
A. C. Higginbotham
H. L. Higgins
N. C. Hightower, Jr.
J. A. Hildes
A. V. Hill (Hon.)
A. Y. Hillier
J. G. Hilton
James Garrett Hilton
H. E. Himwich
W. A. Himwich
H. M. Hines
M. Hines
M. A. Hinrichs
J. C. Hinsey
F. G. Hirsch
F. L. Hisaw
F. A. Hitchcock
H. Hoagland
R. J. Hock
R. Hodes
A. Hodgkin (Hon.)
E. S. Hodgson
P. F. A. Hoefer
B. Hoelscher
E. C. Hoff
H. E. Hoff
B. F. Hoffman
C. A. M. Hogben
A. Hollaender
F. Hollander
W. H. Hollinshead
J. H. Holmes
J. P. Holt
S. W. Hoobler
R. G. Horton
S. M. Horvath
O. Horwitz
B. Houssay (Hon.)
E. Howard
J. E. Howard
D. S. Howell
J. W. Howland
K.-c. Huang
O. L. Huddleston
E. G. Huf
C. B. Huggins
R. A. Huggins
J. Hughes
J. R. Hughes
W. E. Hull
C. C. Hunt
F. R. Hunter
J. Hunter
J. O. Hutchens
C. P. Huttrer
J. E. Hyde
C. Hyman
P. F. Iampietro
C. J. Imig
R. C. Ingraham
W. R. Ingram
L. Irving
A. C. Ivy
J. J. Izquierdo
D. P. Jackson
M. H. Jacobs
E. Jacobson
T. L. Jahn
J. W. Jailer
R. G. Janes
H. D. Janowitz
L. B. Jaques
L. W. Jarcho
H. H. Jasper
K. Jochim
F. H. Johnson
J. A. Johnson
J. L. Johnson
J. R. Johnson
R. E. Johnson
S. A. Johnson
V. Johnson
W. H. Johnson
C. G. Johnston
E. F. Johnston
N. Jolliffe
H. Jonas
H. B. Jones
F. T. Jung
E. C. Jungck
H. Kabat
G. S. Kanter
C.-y. Kao
F. F. Kao
A. Kaplan
H. M. Kaplan
J. G. Kaplan
P. V. Karpovich
S. Katsh
L. N. Katz
J. Kaulbersz
R. A. Kehoe
G. Keighley
N. M. Keith
P. Kellaway
A. D. Keller
R. H. Kellogg
L. S. Kelly
W. Kempner
E. C. Kendall
M. A. Kennard
T. J. Kennedy, Jr.
R. H. Kessler
S. S. Kety
A. Keys
L. G. Kiborn
D. J. Kimeldorf
F. W. Kinard
B. G. King
H. E. King
J. T. King
W. B. Kinter
B. Kisch
C. Kitzinger
C. R. Kleeman
M. Kleiber
I. S. Kleiner
N. Kleitman
D. L. Kline
R. F. Kline
H. M. Klitgaard
H. Kluver
M. H. Knisely
E. Knobil
G. C. Knowlton
C. D. Kochakian
W. P. Koella
G. F. Koepf
K. G. Kohlstaedt
H. I. Kohn
K. Koizumi
W. J. Kolff
S. A. Komarov
T. Koppanyi
I. M. Korr
A. J. Kosman
F. J. Kottke
J. Kramar
B. Kramer
G. L. Kreezer
H. P. Krieger
R. L. Kroc
S. Krop
H. M. Krueger
T. K. Kruse
W. G. Kubicek
S. W. Kuffler
S. Kupfer
A. LaBelle
R. W. Lackey
E. H. Lambert
C. J. Lambertsen
H. Lamport
W. M. Landau
C. Landis
E. M. Landis
M. Landowne
A. M. Lands
A. C. Lane
C. E. Lane
L. L. Langley
E. H. Lanphier
M. G. Larrabee
E. M. Larsen
E. Larson
P. S. Larson
H. D. Lauson
J. H. Lawrence
M. Lawrence
H. Lawson
A. H. Lawton
R. W. Lawton
C. D. Leake
J. H. Leathem
J. A. LeBlanc
L. G. Lederer
C.-C. Lee
D. H. K. Lee
M. O. Lee
R. E. Lee
C. E. Leese
P. G. Lefevre
A. Lein
E. Lepeschkin
M. A. Lessler
J. Y. Lettvin
L. Levenbook
R. Levine
M. F. Levitt
M. N. Levy
A. E. Lewis
B. M. Lewis
D. H. Lewis
J. H. Lewis
L. A. Lewis
C.-l. Li
W. T. Liberson
B. Libet
J. C. R. Licklider
H. S. Liddell
A. L. Lieberman
N. Lifson
G. Liljestrand (Hon.)
J. C. Lilly
R. K. S. Lim
T.-m. Lin
V. F. Lindeman
D. B. Lindsley
G. N. Ling
M. Lipkin
R. W. Lippman
J. M. Little
R. C. Little
R. B. Livingston
C. W. Lloyd
D. P. C. Lloyd
J. B. Loefer
E. R. Loew
C. N. H. Long
G. N. Loofbourrow
L. Lorand
S. Lorber
V. Lorber
R. Lorente de Nó
W. D. Lotspeich
H. S. Loucks
W. E. Love
H. Lowenbach
U. C. Luft
A. A. Luisada
F. D. Lukens
B. J. Luyet
C. P. Lyman
C. A. Maaske
D. M. MacCanon
I. A. Macchi
M. B. Macht
F. C. MacIntosh
E. M. MacKay
I. F. S. Mackay
J. MacLeod
W. O. Maddock
T. B. Magath
D. F. Magee
J. W. Magladery
H. W. Magoun
R. J. Main
F. W. Maire
G. L. Maison
L. I. Malis
J. V. Maloney, Jr.
N. S. R. Maluf
J. F. Manery
F. D. Mann
G. W. Manning
I. A. Manville
J. P. Marbarger
J. E. Markee
J. Markowitz
G. H. Marmont
A. S. Marrazzi
G. Marsh
M. E. Marsh
A. G. Marshak
J. McE. Marshall
L. H. Marshall
W. H. Marshall
A. R. Martin
A. W. Martin, Jr.
S. J. Martin
C. Martinez
E. D. Mason
E. J. Masoro
S. P. Masouredis
G. M. C. Masson
S. A. Matthews
F. W. Maurer
F. R. Mautz
M. Maxfield
M. E. Maxfield
M. H. Maxwell
J. Mayer
H. S. Mayerson
E. L. McCandless
S. McD. McCann
J. D. McCarrell
M. D. McCarthy
E. W. McCheaney
W. S. McClellan
G. P. McCouch
J. W. McCubbin
W. S. McCulloch
F. H. McCutcheon
R. K. McDonald
R. A. McFarland
D. A. McGinty

A. R. McIntyre
P. L. McLain
F. C. McLean
W. C. McNelly
I. McQuarrie
J. Mead
J. Meites
R. M. Melampy
E. S. Mendelson
M. Mendlowitz
G. R. Meneely
H. C. Meng
V. Menkin
A. W. Merrick
J. P. Merrill
F. A. Mettler
D. K. Meyer
R. K. Meyer
W. R. Miles
A. T. Milhorat
A. T. Miller, Jr.
G. A. Miller
J. A. Miller, Jr.
N. Millman
C. A. Mills
D. Minard
A. S. Minot
I. A. Miraky
J. P. Mixner
G. K. Moe
H. Molitor
G. W. Molnar
W. F. H. M. Mommaerts
C. Monge (Hon.)
F. C. Monkhouse
A. V. Montgomery, Jr.
H. Montgomery
D. H. Moore
J. C. Moore
M. F. Morales
L. F. Morehouse
C. F. Morgan
C. T. Morgan
F. A. Morin
R. S. Morison
D. M. Morris
P. R. Morrison
B. Mortimer
C. Moses
W. G. Moss
H. L. Motley
V. B. Mountcastle
C. A. Moyer
S. Mudd
G. H. Mudge
E. E. Muirhead
O. H. Muller
F. J. Mullin
L. J. Mullins
Q. R. Murphy
X. J. Musacchia
J. D. Myers
W. G. Myers
D. Nachmansohn
G. G. Nahas
L. N. Nahum
E. S. Nasset
W. L. Nastuk
H. Necheles
W. D. Neff
D. Nelson
W. O. Nelson
C. Neumann
E. V. Newman
L. B. Nice
J. S. Nicholas
H. C. Nicholson
J. L. Nickerson
M. Nickerson
P. A. Nicoll
L. F. Nims
R. L. Noble
W. K. Noell
T. R. Noonan
D. W. Northup
W. W. Nowinski
J. Nyboer
T. T. Odell, Jr.
E. Ogden
K. N. Ogle
E. A. Ohler
J. R. Oliver
N. S. Olsen
R. A. Olson
W. H. Olson
A. Omachi
D. F. Opdyke
E. T. Oppenheimer
M. J. Oppenheimer
L. A. Orbeli (Hon.)
J. Orloff
O. S. Orth
R. H. Oster
M. I. Osterhout
W. J. V. Osterhout
A. B. Otis
R. R. Overman
C. A. Owen, Jr.
S. E. Owen
D. M. Pace
N. Pace
G. T. Pack
E. Page
E. W. Page
I. H. Page
E. E. Painter-Marcus
E. D. Palmes
J. R. Pappenheimer
C. R. Park
W. M. Parkins
A. K. Parpart
H. O. Parrack
K. E. Paschkis
H. M. Patt
J. L. Patterson, Jr.
T. L. Patterson
H. D. Patton
L. C. Payne
S. M. Peacock
L. J. Pecora
C. N. Peiss
W. G. Penfield
R. Penneys
K. E. Penrod
J. F. Perkins, Jr.
E. R. Perl
P. L. Perlman
J. H. Perlmutt
J. C. Peskin
W. E. Petersen
L. H. Peterson
C. Pfaffmann
J. J. Pfiffner
H. J. Phillips
R. A. Phillips
R. Pick
H. F. Pierce
S. J. Piliero
G. Pincus
I. J. Pincus
J. O. Pinkston
E. A. Pinson
J. Pisanty O.
G. C. Pitts
R. F. Pitts
W. S. Platner
H. Pollack
C. M. Pomerat
W. T. Pommerenke
E. Ponder
J. R. Poppen
E. L. Porter
J. C. Porter
R. W. Porter
R. L. Post
J. M. Posternak
E. J. Poth
E. B. Pratt
K. H. Pribram
H. L. Price
D. F. Proctor
C. L. Prosser
C. B. Puestow
T. N. Pullman
D. P. Purpura
J. P. Quigley
F. H. Quimby
W. Raab
M. S. Raben
E. P. Radford, Jr.
C. Ragan
H. Rahn
N. Rakieten
J. E. Rall
W. Rall
E. P. Ralli
H. J. Ralston
E. R. Ramey
R. W. Ramsey
J. E. Randall
W. C. Randall
R. E. Ranney
D. Rapport
W. J. Rashkind
S. B. Raska
G. C. Rau
I. S. Ravdin
R. W. Rawson
W. O. Read
W. Redisch
E. A. Reed
E. B. Reeve
W. S. Rehm, Jr.
A. F. Reid
M. A. Reid
S. P. Reimann
R. M. Reinecke
E. P. Reineke
J. M. Reiner
W. O. Reinhardt
K. R. Reissmann
A. S. Relman
J. W. Remington
E. M. Renkin
A. E. Renold
M. Reynolds
O. E. Reynolds
S. R. M. Reynolds
P. Reznikoff
R. Rhines
J. E. Rhoads
H. V. Rice
D. W. Richards, Jr.
O. W. Richards
R. K. Richards
A. W. Richardson
C. P. Richter
H. T. Ricketts
P. Rieser
L. A. Riggs
R. L. Riley
G. C. Ring
D. M. Rioch
J. S. Robb
J. T. Roberts
K. E. Roberts
S. Roberts
J. S. Robertson
S. Robinson
T. W. Robinson
F. S. Robscheit-Robbins
M. Rockstein
K. Rodahl
S. Rodbard
K. D. Roeder
W. G. Rohse
D. T. Rolfe
A. L. Romanoff
E. B. Romanoff
R. R. Ronkin
A. Roos
R. W. Root
W. S. Root
J. C. Rose
J. E. Rose
A. Rosenblueth
G. Rosenfeld
R. H. Rosenman
S. R. Rosenthal
B. B. Ross
R. S. Ross
H. H. Rostorfer
G. M. Roth
J. L. d'A. Roth
L. W. Roth
I. Rothchild
A. Rothstein
F. J. W. Roughton (Hon.)
B. B. Rubenstein
T. C. Ruch
D. O. Rudin
G. G. Rudolph
C. H. W. Ruhe
R. F. Rushmer
A. Ruskin
J. A. Russell
A. H. Ryan
J. Sacks
P. F. Salisbury
S. C. Samaras
P. Samet
J. J. Sampson
F. E. Sampson, Jr.
A. Sandow
D. J. Sandweiss
L. A. Sapirstein
F. Sargent, II
S. J. Sarnoff
O. W. Sartorius
L. J. Saul
F. J. Saunders
C. H. Sawyer
M. E. MacK. Sawyer
W. H. Sawyer
G. Sayers
R. E. Scantlebury
K. E. Schaefer
W. B. Schallek
R. W. Schayer
B. T. Scheer
A. M. Scher
M. J. Schiffrin
J. Schiller
J. U. Schlegel
B. H. Schlomovitz
C. F. Schmidt
C. R. Schmidt
B. Schmidt-Nielsen
K. Schmidt-Nielsen
F. O. Schmitt
O. H. Schmitt
C. L. Schneider
J. A. Schneider
L. H. Schneyer
H. W. Schoenborn
G. M. Schoepfle
P. F. Scholander
G. E. Schreiner
H. A. Schroeder
I. L. Schwartz
N. B. Schwartz
W. B. Schwartz
M. Schweizer
H. Schwerma
D. Scott, Jr.
J. C. Scott
V. B. Scott
W. W. Scott
R. O. Scow
N. S. Scrimshaw
G. R. Seaman
W. H. Seegers
M. H. Seevers
E. E. Selkurt
W. A. Selle
A. L. Sellers

E. A. Sellers
H. Selye
R. E. Semple
J. Sendroy, Jr.
M. W. Seraydarian
J. W. Severinghaus
R. W. Sevy
O. W. Shadle
A. M. Shanes
J. A. Shannon
H. Shapiro
L. Share
J. C. Shaw
H. Shay
C. Sheard
D. Sheehan
M. C. Shelesnyak
W. B. Shelley
R. H. Shepard
C. W. Sheppard
F. G. Sherman
S. Sherry
T. C. Sherwood
L. B. Shettles
C. W. Shilling
R. A. Shipley
R. E. Shipley
S. Shlaer
N. W. Shock
F. J. M. Sichel
A. A. Siebens
E. B. Sigg
H. Silvette
D. H. Simmons
E. Simonson
M. E. Simpson
H. O. Singher
J. H. Sirota
I. W. Sizer
W. Sleator, Jr.
D. C. Smith
D. E. Smith
D. J. Smith
E. A. Smith
E. L. Smith
F. Smith
H. P. Smith
H. W. Smith
J. A. Smith
J. J. Smith
J. R. Smith
P. W. Smith
R. E. Smith
W. K. Smith
W. W. Smith
W. J. Snape
J. G. Snedecor
R. S. Snider
F. F. Snyder
S. S. Sobin
A. L. Soderwall
L. Sokoloff
A. K. Solomon
R. R. Sonnenschein
S. Soskin
T. Spaet
C. R. Spealman
H. Specht
R. S. Speirs
M. P. Spencer
R. W. Sperry
S. S. Spicer
E. A. Spiegel
S. Spiegelman
J. D. Spikes
J. J. Spitzer
H. J. Spoor
J. M. Sprague
G. B. Spurr
R. D. Squires
R. W. Stacy
J. Stamler
J. N. Stannard
L. Stark
I. Starr
G. W. Stavraky
E. A. Stead, Jr.
J. M. Steele
M. Stefanini
F. R. Steggerda
S. E. Steiman
S. N. Stein
H. B. Steinbach
A. H. Steinhaus
S. S. Stevens
J. A. F. Stevenson
D. R. Stewart
P. A. Stewart
J. C. Stickney
T. J. B. Stier
E. U. Still
J. W. Still
A. M. Stoll
W. E. Stone
H. C. Struck
H. Strughold
P. D. Sturkie
J. W. Stutzman
E. E. Suckling
F. A. Sunahara
A. Surtshin
G. F. Sutherland
H. J. C. Swan
R. C. Swan, Jr.
H. G. Swann
H. M. Sweeney
M. N. Swift
W. W. Swingle
J. F. Sykes
C. M. Szego
A. G. Szent-Györgyi
J. Szepsenwol
J. V. Taggart
T. N. Tahmisian
M. L. Tainter
S. A. Talbot
R. V. Talmage
P. C. Tang
I. Tasaki
A. N. Taylor
C. B. Taylor
H. L. Taylor
N. B. Taylor
R. D. Taylor
R. E. Taylor
R. M. Taylor
R. D. Teasdall
R. D. Templeton
S. M. Tenney
H. M. Tepperman
J. Tepperman
C. A. Terzuolo
J. S. Thatcher
R. Thauer
P.-O. Theman
G. J. Thomas
J. E. Thomas
D. D. Thompson
J. D. Thomson
J. F. Thomson
G. W. Thorn
S. R. Tipton
R. Tislow
L. Tobian, Jr.
J. M. Tobias
L. M. Tocantins
J. E. P. Toman
J. F. Tomashefski
L. A. Toth
A. L. Towe
S. S. Tower
L. E. Travis
H. J. Trumit
R. D. Tschirgi
C. tum Suden
A. R. Tunturi
W. W. Tuttle
D. B. Tyler
G. Ungar
K. R. W. Unna
M. Upton
J. C. Vanatta
H. B. van Dyke
A. Van Harreveld
E. J. Van Liere
L. Van Middlesworth
G. Van Wagenen
J. T. Velardo
M. Verzeano
R. Villarreal
F. E. Visscher
M. B. Visscher
K. von Frisch (Hon.)
K. N. von Kaulla
N. J. Wade
I. H. Wagman
H. G. Wagner
G. E. Wakerlin
K. G. Wakim
W. W. Walcott
J. M. Waldron
A. E. Walker
S. M. Walker
P. D. Wall
M. Waiser
O. S. Walters
G. H. Wang
S. C. Wang
O. H. Wangensteen
A. A. Ward, Jr.
G. F. Warner
H. R. Warner
J. V. Warren
K. Wasserman
D. M. Watkin
J. B. Watson
D. F. Waugh
J. T. Weam
P. Webb
R. Wegria
C. K. Weichert
V. L. Weimar
E. G. Weir
A. K. Weiss
H. S. Weiss
P. A. Weiss
C. B. Weld
H. S. Wells
J. H. Welsh
L. G. Welt
J. M. Werle
G. E. Wertenberger
N. T. Werthessen
L. G. Wesson, Jr.
C. D. West
B. A. Westfall
R. E. Weston
H. Wheelon
A. G. White
H. L. White
R. W. Whitehead
W. V. Whitehorn
J. E. Whitney
J. L. Whittenberger
M. P. Wiedeman
F. J. Wiercinski
C. A. G. Wiersma
H. C. Wiggers
H. S. Wigodsky
C. G. Wilber
K. M. Wilbur
W. S. Wilde
C. M. Wilhelmj
C. M. Williams
M. F. Williams
M. H. Williams, Jr.
W. L. Williams
T. L. Willmon
J. H. Wills
J. Walter Wilson
John W. Wilson
L. P. Wilson
V. J. Wilson
W. L. Wilson
C. V. Winder
W. F. Windle
C. A. Winter
A. V. Wolf
S. Wolf
H. G. Wolff
S. H. Wollman
L. F. Wolterink
H. Y. C. Wong
E. H. Wood
J. W. Woodbury
L. A. Woodbury
R. A. Woodbury
A. H. Woodcock
E. R. Woodward
C. N. Woolsey
S. B. Wortis
C. I. Wright
E. B. Wright
G. W. Wright
P. A. Wright
V. J. Wulff
C. C. Wunder
L. C. Wyman
W. Wynn
F. E. Yates
W. B. Youmans
A. C. Young
T.-F. Yu
J. A. Zapp, Jr.
M. X. Zarrow
K. L. Zierler
D. B. Zilversmit
R. E. Zirkle
M. B. Zucker
B. W. Zweifach
R. L. Zwemer

RETIRED MEMBERS

W. H. F. Addison
E. C. Albritton
F. M. Allen
W. C. Alvarez
S. Amberg
A. Arkin
H. G. Armstrong
R. Ashman
J. C. Aub
G. Bachmann
G. A. Baitsell
J. P. Baumberger
O. Bergeim
R. H. Beutner
N. R. Blatherwick
W. R. Bloor
H. C. Bradley
S. W. Britton
E. D. Brown
E. Bulatao
T. M. Carpenter
H. W. Chalkley
E. R. Clark
J. H. Clark
M. E. Collett
G. R. Cowgill
E. A. K. Culler
R. S. Cunningham
P. M. Dawson
A. W. Downs
E. F. DuBois
D. J. Edwards
J. Erlanger
H. M. Evans
J. A. E. Eyster
G. Fahr
M. P. Fitzgerald
E. B. Forbes
H. S. Forbes
E. M. K. Geiling
T. S. Githens
M. H. Givens

F. R. Griffith, Jr.
C. Gruber
A. Gulick
C. C. Guthrie
H. H. Haggard
W. Hale
O. G. Harne
R. G. Harrison
C. G. Hartman
F. A. Hartman
E. N. Harvey
F. A. Hellebrandt
C. J. Herrick
C. L. Hess
H. G. O. Holck
D. Hooker
R. G. Hoskins
P. E. Howe
D. E. Jackson
K. K. Jones
F. P. Knowlton
E. B. Krumbhaar
M. M. Kunde
P. D. Lamson
K. S. Lashley
H. Laurens
W. H. Lewis
A. E. Livingston
E. Lodholz
L. Loeb
R. O. Loebel
B. R. Lutz
D. I. Macht
F. C. Mann
D. Marine
E. K. Marshall, Jr.
J. W. Mavor
J. F. McClendon
H. A. McGuigan
W. J. Meek
M. L. Menten
A. E. Meyer
F. R. Miller
A. R. Moore
V. H. K. Moorhouse
S. Morgulis
J. Murlin
E. L. Opie
S. E. Pond
F. H. Pratt
A. C. Redfield
C. I. Reed
A. N. Richards
O. Riddle
G. C. Robinson
F. T. Rogers
J. M. Rogoff
G. B. Roth
L. G. Rowntree
E. Sachs
E. L. Scott
C. A. Smith
P. E. Smith
C. D. Snyder
T. Sollmann
O. O. Stoland
S. Tashiro
C. Voegtlin
F. W. Weymouth
G. H. Whipple
C. J. Wiggers
R. M. Wilder
D. W. Wilson
A. E. Woodward
R. Wulzen
I. T. Zeckwer

American Society of Biological Chemists

ACTIVE MEMBERS

L. D. F. Abbott, Jr.
L. L. Abell
S. Abraham
A. Abrams
R. Abrams
H. A. Abramson
W. W. Ackermann
E. Adams
M. Adams
E. H. Ahrens, Jr.
A. A. Albanese
H. G. Albaum
R. A. Alberty
G. Alderton
B. Alexander
R. B. Alfin-Slater
S. G. A. Alivisatos
F. W. Allen
J. B. Allison
H. J. Almquist
K. I. Altman
A. M. Altschul
B. N. Ames
S. Ames
M. Anchel
M. A. Andersch
C. E. Anderson
E. P. Anderson
L. Anderson
R. J. Anderson
C. B. Anfinsen, Jr.
H. S. Anker
S. Ansbacher
W. P. Anslow, Jr.
M. L. Anson
R. M. Archibald
M. D. Armstrong
W. D. Armstrong
D. I. Arnon
L. E. Arnow
S. Aronoff
C. Artom
C. F. Asenjo
J. Ashmore
G. G. Ashwell
L. Atkin
D. E. Atkinson
J. Awapara
A. E. Axelrod
B. Axelrod
J. E. Bacher
C. Bachman
E. Baer
H. D. Baernstein
B. Baggett
C. G. Baker
N. Baker
R. L. Baldwin
M. E. Balis
E. G. Ball
C. E. Ballou
A. K. Balls
R. S. Bandurski
R. K. Barclay
H. A. Barker
F. W. Barnes, Jr.
R. H. Barnes
C. P. Barnum, Jr.
H. W. Barrett
G. T. Barry
G. R. Bartlett
P. D. Bartlett
D. H. Basinski
R. W. Bates
C. A. Baumann
E. F. Beach
B. W. Beadle
H. H. Beard
J. R. Beaton
W. S. Beck
R. R. Becker
R. F. Beers, Jr.
E. Beerstecher, Jr.
H. Beevers
R. W. Begg
W. T. Beher
J. A. Behre
O. K. Behrens
H. Beinert
P. H. Bell
A. Bendich
R. Benesch
R. E. Benesch
E. L. Bennett
M. A. Bennett
A. A. Benson
R. Bentley
C. P. Berg
P. Berg
W. Bergmann
P. Bernfeld
F. Bernheim
I. A. Bernstein
O. A. Bessey
S. P. Bessman
C. H. Best
R. M. Bethke
M. Bier
C. E. Bills
F. C. Bing
F. Binkley
S. B. Binkley
O. Bird
S. M. Birnbaum
F. E. Bischoff
A. L. Black
S. Black
N. F. Blau
K. Bloch
R. J. Block
W. D. Block
B. Bloom
R. M. Bock
O. Bodansky
A. Bolliger
D. M. Bonner
J. Bonner
R. W. Bonsnes
S. L. Boating
L. E. Booher
I. J. U. Boone
A. N. Booth
E. Borek
H. Borsook
P. A. Bott
J. Bourdillon
M. R. Bovarnick
D. E. Bowman
G. E. Boxer
M. J. Boyd
P. D. Boyer
H. L. Bradlow
R. O. Brady
G. Brawerman
F. G. Brazda
C. R. Brewer
D. R. Briggs
M. Brin
N. G. Brink
A. F. Brodie
B. B. Brodie
H. P. Broquist
D. H. Brown
G. B. Brown
J. B. Brown
R. K. Brown
T. C. Bruice
D. L. Buchanan
J. M. Buchanan
E. Bueding
M. V. Buell
H. Bull
H. B. Burch
D. Burk
J. J. Burns
G. O. Burr
W. W. Burr, Jr.
R. H. Burris
R. M. Burton
H. Busch
G. C. Butler
J. S. Butts
R. U. Byerrum
W. M. Cahill
F. A. Cajori
M. L. Caldwell
D. B. Calvin
M. Calvin
M. N. Camien
L. L. Campbell, Jr.
E. S. Canellakis
R. K. Cannan
G. L. Cantoni
R. Caputto
E. B. Carmichael
F. H. Carpenter
C. W. Carr
K. K. Carroll
C. Carruthers
S. F. Carson
M. E. Carsten
C. E. Carter
H. E. Carter
G. F. Cartland
H. G. Cassidy
W. O. Caster
L. F. Cavalieri
J. J. Ceithaml
W. D. Celmer
L. R. Cerecedo
B. Chance
J. P. Chandler
A. Chanutin
F. C. Charalampous
E. Chargaff
J. Charney
A. M. Chase
V. H. Cheldelin
S. S. Chernick
F. P. Chinard
B. F. Chow
H. N. Christensen
A. A. Christman
J. A. Cifonelli
I. Clark
H. T. Clarke
R. E. Clegg
R. D. Coghill
P. P. Cohen
S. Cohen
S. L. Cohen
S. S. Cohen
Melvin Cohn
Mildred Cohn
W. E. Cohn
R. D. Cole

H. B. Collier
J. B. Collip
S. P. Colowick
C. L. Comar
B. Commoner
P. Condliffe
J. G. Coniglio
E. E. Conn
W. V. Consolazio
T. B. Coolidge
M. J. Coon
C. Cooper
J. H. Copenhaver
C. F. Cori
R. C. Corley
W. E. Cornatzer
J. R. Couch
R. A. Coulson
G. R. Cowgill
R. W. Cowgill
G. L. Cox
W. M. Cox, Jr.
L. C. Craig
C. F. Crampton
D. I. Crandall
F. L. Crane
R. K. Crane
F. A. Csonka
L. S. Cunningham
L. W. Cunningham
G. L. Curran
J. M. Curtis
F. S. Daft
H. Dam
W. B. Dandliker
L. J. Daniel
I. S. Danielson
W. J. Darby
D. C. Darrow
B. F. Daubert
R. E. Davies
B. D. Davis
G. K. Davis
N. C. Davis
C. R. Dawson
H. G. Day
P. L. Day
F. E. Deatherage
F. De Eds
C. A. Dekker
C. C. Delwiche
E. A. Delwiche
R. H. De Meio
R. D. De Moss
O. F. Denstedt
E. C. DeRenzo
V. Deulofeu
H. F. Deutsch
V. C. Dewey
S. R. Dickman
L. S. Dietrich
D. B. Dill
R. T. Dillon
Z. Dische
K. Dittmer
V. M. Doctor
D. G. Doherty
E. A. Doisy, Sr.
E. A. Doisy, Jr.
M. I. Dolin
A. Dorfman
R. I. Dorfman
J. R. Doty
P. M. Doty
M. Doudoroff
A. L. Dounce
D. L. Drabkin
W. Drell
H. E. Dubin
J. W. Dubnoff
E. L. Duggan
F. W. Dunn
M. S. Dunn
E. L. Durrum

J. D. Dutcher
V. du Vigneaud
H. M. Dyer
D. D. Dziewiatkowski
H. Eagle
R. E. Eakin
H. C. Eckstein
H. Edelhoch
H. A. Eder
J. T. Edsall
M. R. Ehrenstein
H. J. Eichel
L. Eichelberger
J. J. Eiler
F. Eisenberg, Jr.
M. A. Eisenberg
A. J. Eisenman
R. C. Elderfield
G. B. Elion
E. Ellenbogen
K. A. C. Elliott
W. H. Elliott
N. R. Ellis
D. Elson
C. A. Elvehjem
D. Elwyn
N. D. Embree
L. L. Engel
S. Englard
J. English, Jr.
B. F. Erlanger
E. A. Evans, Jr.
D. F. Eveleth
M. R. Everett
C. F. Failey
D. Fairbairn
J. L. Fairley, Jr.
F. W. Fales
E. Farber
M. Fay
R. E. Feeney
P. Feigelson
D. S. Feingold
R. N. Feinstein
J. D. Ferry
R. M. Ferry
H. L. Fevold
J. B. Field
K. Fink
R. M. Fink
E. H. Fischer
M. A. Fischer
E. H. Fishberg
A. M. Fisher
H. H. Fisher
W. H. Fishman
M. Flavin
G. A. Fleisher
L. B. Flexner
E. V. Flock
N. F. Floyd
E. H. Flynn
P. J. Fodor
J. Folch
J. E. Folk
K. Folkers
T. D. Fontaine
J. C. Forbes
L. S. Fosdick
J. F. Foster
J. W. Foster
J. J. Fox
S. W. Fox
H. L. Fraenkel-Conrat
I. D. Frantz
A. H. Free
C. S. French
D. French
A. W. Frenkel
J. Fried
F. Friedberg
T. E. Friedemann
E. Frieden
E. H. Frieden

M. E. Friedkin
W. R. Frisell
C. E. Frohman
D. V. Frost
J. S. Fruton
S.-C. J. Fu
D. K. Fukushima
M. Fuld-Conderman
R. F. Furchgott
B. W. Gabrio
O. H. Gaebler
H. Graffron
E. M. Gal
T. F. Gallagher
W. D. Gallup
A. W. Galston
R. L. Garner
J. B. Garst
O. Gawron
W. F. Geddes
C. L. Gemmill
P. George
J. Gergely
H. Gest
M. Gibbs
D. M. Gibson
J. B. Gilbert
C. Gilvarg
E. C. Gjessing
D. Glick
D. R. Goddard
W. F. Goebel
M. Goettsch
A. R. Goldfarb
D. S. Goldman
P. D. Goldsworthy
D. A. Goldthwait
E. Goldwasser
I. Goodman
W. G. Gordon
L. Gorini
W. A. Gortner
H. Goss
B. S. Gould
R. G. Gould
S. Graff
C. E. Graham
S. Granick
R. L. Grant
D. E. Green
H. Green
D. M. Greenberg
G. R. Greenberg
L. D. Greenberg
C. H. Greene
J. P. Greenstein
J. D. Gregory
A. C. Griffin
W. H. Griffith
S. Grisolia
E. G. Gross
N. B. Guerrant
G. M. Guest
I. C. Gunsalus
F. R. Gurd
S. Gurin
E. L. Gustus
A. B. Gutman
H. R. Gutmann
P. György
J. R. Haag
E. Haas
M. H. Hack
L. P. Hager
D. D. Hagerman
H. W. Haggard
F. E. Hahn
W. J. Haines
R. H. Hall
W. K. Hall
B. L. K. Hamilton
P. B. Hamilton
T. S. Hamilton
D. J. Hanahan

D. B. Hand
P. Handler
C. S. Hanes
M. E. Hanke
M. T. Hanke
L. V. Hankes
R. G. Hansen
I. Harary
H. A. Harbury
R. N. Harger
B. K. Harned
A. E. Harper
H. A. Harper
W. F. Harrington
D. L. Harris
M. Harris
P. L. Harris
A. F. Hartman
W. Z. Hassid
A. B. Hastings
N. Haugaard
F. Haurowitz
W. Hausmann
F. L. Haven
P. B. Hawk
W. W. Hawkins
O. Hayaishi
M. Hayano
E. E. Hays
F. C. Heagy
C. Heidelberger
M. R. Heinrich
E. Heinz
L. Hellerman
O. M. Helmer
L. M. Henderson
R. W. Hendler
L. A. Heppel
R. M. Herbst
R. M. Herriott
E. L. Hess
G. P. Hess
W. C. Hess
H. H. Hiatt
S. W. Hier
R. L. Hill
R. M. Hill
C. H. W. Hirs
E. Hirschberg
H. Hirschmann
D. I. Hitchcock
G. H. Hitchings
M. B. Hoagland
H. D. Hoberman
F. L. Hoch
H. Hoch
R. M. Hochster
R. C. Hockett
H. C. Hodge
W. S. Hoffman
K. Hofmann
B. H. J. Hofstee
J. F. Hogg
L. E. Hokin
J. T. Holden
F. Hollander
V. P. Hollander
R. W. Holley
R. T. Holman
J. O. Holmes
L. E. Holt, Jr.
I. M. Hoobler
S. R. Hoover
B. L. Horecker
M. J. Horn
N. H. Horowitz
B. N. Horwitt
M. K. Horwitt
R. D. Hotchkiss
E. E. Howe
S. F. Howell
S.-L. Hsia
H. T. Huang
R. H. Hubbard

F. M. Huennekens
J. W. Huff
M. N. Huffman
J. S. Hughes
W. L. Hughes
J. P. Hummel
F. E. Hunter, Jr.
R. B. Hurlbert
J. Hurwitz
B. L. Hutchings
M. Ikawa
L. L. Ingraham
V. Iob
E. M. Irvin
J. L. Irvin
G. W. Irving, Jr.
H. A. Itano
R. W. Jackson
W. B. Jacoby
B. J. Jandorf
E. F. Jansen
R. W. Jeanloz
W. P. Jencks
R. Jenness
E. V. Jensen
H. F. Jensen
M. A. Jesaitis
B. C. Johnson
M. J. Johnson
R. E. Johnson
R. M. Johnson
M. W. Johnston
R. B. Johnston
J. H. Jones
M. E. Jones
N. R. Joseph
M. A. Joslyn
T. H. Jukes
E. Juni
E. A. Kabat
P. Kaesberg
H. M. Kalckar
R. E. Kallio
G. Kalnitsky
M. D. Kamen
H. Kamin
N. O. Kaplan
M. L. Karnovsky
M. Karshan
F. Karush
B. Kassell
J. Katz
P. A. Katzman
S. Kaufman
E. R. M. Kay
L. A. Kazal
E. B. Keamey
E. O. Keiles
E. B. Keller
E. P. Kennedy
J. C. Kereszttesy
S. E. Kerr
Z. I. Kertesz
A. S. Keston
A. Keys
H. G. Khorana
A. C. Kibrick
G. W. Kidder
R. K. Kielley
W. W. Kielley
M. W. Kies
J. R. Kimmel
C. G. King
T. E. King
J. H. Kinoshita
V. E. Kinsey
E. R. Kirch
J. E. Kirk
P. L. Kirk
S. Kirkwood
S. Kit
S. J. Klebanoff
J. R. Klein
R. Klemperer
I. M. Klotz
C. A. Knight
W. E. Knox
A. L. Koch
C. D. Kochakian
W. F. Kocholaty
A. E. Koehler
V. L. Koenig
R. E. Koeppe
H. Koffler
E. D. Korn
A. Komberg
D. E. Koshland, Jr.
L. M. Kozloff
A. R. Krahl
M. E. Krahl
B. Kramer
L. O. Krampitz
R. F. Krause
W. E. Krauss
E. G. Krebs
W. A. Krehl
T. H. Kritchevsky
H. D. Kruse
S. A. Kuby
K. A. Kuiken
M. H. Kuizenga
E. Kun
H. G. Kunkel
H. O. Kunkel
D. W. Kupke
A. Kurtz
A. C. Kuyper
G. C. Kyker
B. N. La Du, Jr.
A. Lajtha
J. P. Lambooy
J. O. Lampen
R. G. Langdon
W. H. Langham
W. D. Langley
H. A. Lardy
J. Larner
B. L. Larson
H. W. Larson
M. Laskowski
M. Laskowski, Jr.
M. A. Lauffer, Jr.
E. P. Laug
P. S. Lavik
T. F. Lavine
I. G. Leder
N. D. Lee
A. L. Lehninger
L. F. Leloir
J. R. Leonards
G. A. Le Page
S. Lepkovsky
A. B. Lerner
S. Levey
L. Levin
H. Levine
V. E. Levine
L. Levintow
M. Levitz
M. Levy
G. T. Lewis
J. C. Lewis
U. J. Lewis
M. V. L'Heureux
C. H. Li
H. C. Lichstein
I. Lieberman
S. Lieberman
I. E. Liener
H. D. Lightbody
A. Lindenbaum
H. Lineweaver
K. P. Link
A. Linker
F. J. Lionetti
F. Lipmann
M. A. Lipton
H. N. Little
G. Litwack
F. A. Loewus
R. B. Loftfield
M. A. Logan
M. E. Lombardo
I. M. London
C. N. H. Long
H. E. Longenecker
B. B. Longwell
W. F. Loomis
J. M. Looney
L. Lorand
H. S. Loring
L. H. Louis
B. W. Low
O. H. Lowry
C. C. Lucas
G. H. W. Lucas
J. M. Luck
S. Ludewig
R. Lumry
W. O. Lundberg
H. P. Lundgren
C. M. Lyman
W. K. Maas
D. W. MacCorquodale
D. L. MacDonald
N. S. MacDonald
D. A. MacFadyen
C. G. Mackenzie
J. B. Mackenzie
G. Mackinney
B. Mackler
F. L. MacLeod
R. A. MacLeod
R. MacVicar
G. D. Maengwyn-Davies
B. Magasanik
H. R. Mahler
R. T. Major
G. F. Maley
M. F. Mallette
E. B. Man
H. G. Mandel
I. Mandl
G. H. Mangun
G. V. Marinetti
P. A. Marks
J. Marmur
J. Marsh
L. M. Marshall
G. J. Martin
W. Marx
H. L. Mason
H. S. Mason
Merle Mason
Morton F. Mason
V. Massey
M. B. Mathews
J. C. Mathies
V. R. Mattox
F. H. Matteson
M. E. Maver
E. S. Maxwell
D. T. Mayer
A. Mazur
C. M. McCay
K. P. McConnell
R. H. McCoy
F. G. McDonald
H. J. McDonald
W. McElroy
W. D. McElroy
W. D. McFarlane
R. W. McGilvery
E. W. McHenry
F. C. McIntire
R. W. McKee
H. McKennis, Jr.
J. M. McKibbin
A. D. McLaren
I. R. McManus
T. L. McMeekin
W. H. McShan
J. F. Mead
G. Medes
J. W. Mehl
A. H. Mehler
A. Meister
J. B. Melchior
R. C. Mellors
D. Melnick
D. B. Melville
B. Mendel
R. B. Merrifield
E. T. Mertz
D. E. Metzler
A. S. Meyer
C. E. Meyer
K. Meyer
M. Michaelis
H. O. Michel
O. Mickelsen
B. B. Migicovsky
E. Mihalyi
W. E. Militzer
B. F. Miller
E. C. Miller
G. L. Miller
J. A. Miller
L. Miller
L. L. Miller
O. N. Miller
Z. B. Miller
C. A. Mills
R. C. Mills
A. E. Mirsky
H. K. Mitchell
C. Mitoma
K. Moldave
W. F. H. M. Mommaerts
R. Montgomery
S. Moore
M. Morehouse
S. A. Morell
J. F. Morgan
H. P. Morris
D. B. Morrison
M. Morrison
P. R. Morrison
M. Morse
E. H. Moubach
J. W. Moulder
L. A. Mounter
A. L. Moxon
H. S. Moyed
G. C. Mueller
D. J. Mulford
J. W. Mull
P. L. Munson
E. Muntwyler
J. A. Muntz
J. Muus
D. Nachmansohn
V. A. Najjar
H. I. Nakada
T. P. Nash, Jr.
A. Nason
S. Natelson
J. B. Neilands
C. H. Neilson
E. M. Nelson
N. Nelson
W. L. Nelson
W. F. Neuman
H. Neurath
I. Neuwirth
R. W. Newburgh
P. Newmark
C. A. Nichol
W. J. Nickerson
C. G. Niemann
A. Nisonoff
L. Noda
H. Noll
F. F. Nord
L. C. Norris
W. P. Norris

J. H. Northrop
G. D. Novelli
A. B. Novikoff
J. F. Nyc
F. W. Oberst
S. Ochoa
B. L. O'Dell
P. Oesper
M. J. Oesterling
E. L. Oginsky
R. Okey
H. S. Olcott
J. J. Oleson
N. S. Olsen
J. A. Olson
O. E. Olson
R. E. Olson
J. L. Oncley
J. C. Opsahl
A. A. Ormsby
J. M. Orten
A. E. Osterberg
I. H. Page
J. K. Palmer
M. C. Pangborn
E. Papageorge
A. M. Pappenheimer, Jr.
A. B. Pardee
C. R. Park
J. H. Park
J. T. Park
E. K. Patterson
E. L. Patterson
W. I. Patterson
M. F. Paul
J. H. Pazur
W. H. Pearlman
O. H. Pearson
P. B. Pearson
J. D. Perkinson, Jr.
G. E. Perlmann
M. L. Petermann
T. Peters, Jr.
E. A. Peterson
R. D. Peterson
J. J. Pfiffner
P. H. Phillips
H. B. Pierce
J. G. Pierce
K. A. Piez
W. W. Pigman
L. O. Pilgeram
G. B. Pinchot
G. W. E. Plaut
W. J. Polglase
A. Pope
E. A. Popenoe
C. C. Porter
J. W. Porter
R. L. Potter
V. R. Potter
M. H. Power
P. W. Preisler
D. Pressman
J. M. Price
V. E. Price
W. H. Prusoff
L. I. Pugsley
E. W. Putman
F. W. Putnam
F. W. Quackenbush
M. L. Qunife
J. H. Quastel
A. J. Quick
M. Rabinovitz
J. C. Rabinowitz
J. L. Rabinowitz
J. R. Rachele
E. Racker
N. S. Radin
M. E. Rafelson, Jr.
N. W. Rakestraw
L. O. Randall
S. Rapoport
M. M. Rapport
S. Ratner
J. M. Ravel
A. L. Raymond
L. J. Reed
R. E. Reeves
H. Reich
J. C. Reid
J. G. Reinhold
R. Reiser
F. J. Reithel
C. N. Remy
A. G. Renfrew
C. Ressler
F. M. Richards
D. A. Richert
B. Riegel
C. Riegel
A. F. Riggs
T. R. Riggs
D. Rittenberg
E. Roberts
S. Roberts
W. van B. Robertson
E. Robins
H. W. Robinson
R. O. Roblin, Jr.
E. Roboz
L. B. Rockland
F. L. Rodkey
J. H. Roe
P. M. Roll
E. Ronzoni
C. S. Rose
I. A. Rose
W. C. Rose
S. Roseman
H. R. Rosenberg
I. Rosenfeld
R. S. Rosenfeld
H. Rosenkrantz
H. L. Rosenthal
O. Rosenthal
R. J. Rossiter
J. S. Roth
P. W. K. Rothemund
M. Rothstein
G. Rouser
J. I. Routh
S. H. Rubin
H. Rudney
W. R. Ruegamer
R. J. Rutman
W. J. Rutter
H. Z. Sable
H. Sachs
B. Sacktor
M. Sahyun
W. Sakami
H. J. Sallach
W. D. Salmon
K. Salomon
P. Saltman
L. T. Samuels
D. R. Sanadi
A. San Pietro
H. P. Sarett
G. H. Satterfield
H. E. Sauberlich
F. G. K. Savard
H. J. Saz
H. K. Schachman
N. K. Schaffer
O. Schales
R. W. Schayer
A. H. Schein
J. R. Schenck
H. A. Scheraga
S. Schiller
M. Schlamowitz
F. Schlenk
K. Schmid
E. G. Schmidt
G. Schmidt
L. H. Schmidt
J. J. Schneider
W. C. Schneider
J. Schubert
W. J. Schubert
M. P. Schulman
J. Schultz
M. O. Schultze
S. Schwartz
K. Schwarz
R. S. Schweet
B. S. Schweigert
C. E. Schwerdt
G. W. Schwert
S. Schwimmer
D. B. McN. Scott
E. M. Scott
M. L. Scott
J. V. Scudi
W. H. Sebrell, Jr.
W. H. Seegers
F. B. Seibert
S. Seifter
H. M. Sell
J. Sendroy, Jr.
M. G. Sevag
E. L. Severinghaus
J. Shack
P. A. Shaffer
R. E. Shank
E. Shaw
K. N. F. Shaw
M. J. Shear
A. L. Sheffner
D. Shemin
F. Sheppard
G. Y. Shinowara
W. Shive
W. W. Shreeve
S. Shulman
L. Shuster
P. Siekevitz
R. H. Silber
M. Silverman
S. Simmonds
E. J. Simon
D. G. Simonsen
M. V. Simpson
F. M. Sinex
S. A. Singal
L. Singer
S. J. Singer
T. P. Singer
R. L. Sinsheimer
I. W. Sizer
C. G. Skinner
J. T. Skinner
H. E. Skipper
F. Skoog
M. W. Slein
N. H. Sloane
H. A. Sloviter
A. H. Smith
E. L. Smith
E. R. B. Smith
F. G. Smith
G. N. Smith
H. W. Smith
L. Smith
L. I. Smith
P. K. Smith
C. V. Smythe
E. E. Snell
J. E. Snoke
A. E. Sobel
H. A. Sober
H. H. Sobotka
M. Sonenberg
M. Soodak
S. Sorof
T. L. Sourkes
W. M. Sperry
M. Spiegel-Adolf
J. Spizizen
D. B. Sprinson
E. R. Stadtman
T. C. Stadtman
M. A. Stahmann
R. Y. Stanier
W. M. Stanley
P. G. Stansly
E. Staple
F. J. Stare
Z. Stary
A. Staub
H. E. Stavely
G. Stearns
R. Steele
S. L. Steelman
W. H. Stein
D. Steinberg
J. Steinhardt
H. G. Steinman
J. A. Stekol
J. R. Stern
DeW. Stetten, Jr.
M. R. Stetten
C. M. Stevens
B. F. Stimmel
C. C. Stock
E. L. R. Stokstad
D. Stone
G. C. H. Stone
E. H. Stotz
M. Strassman
H. J. Strecker
B. L. Strehler
D. R. Strength
K. P. Strickland
C. F. Strittmatter
J. L. Strominger
F. M. Strong
P. K. Stumpf
W. H. Summerson
F. W. Sunderman
D. MacN. Surgenor
E. W. Sutherland, Jr.
W. B. Sutton
M. A. Swanson
W. W. Swanson
M. L. Sweat
L. Swell
M. E. Swendseid
R. W. Swick
H. E. Swim
A. Szent-Györgyi
A. G. Szent-Györgyi
H. Tabor
P. Talalay
H. H. Tallan
S. W. Tanenbaum
H. L. A. Tarr
H. Tarver
E. L. Tatum
H. Tauber
A. Taurog
F. A. Taylor
J. F. Taylor
T.-t. Tchen
D. M. Tennent
L. J. Teply
C. Terner
S. J. Thannhauser
S. A. Thayer
A. W. Thomas
L. E. Thomas
W. R. Thompson
D. L. Thomson
C. B. Thorne
H. C. Tidwell
R. S. Tipson
E. O. Titus
H. W. Titus
W. R. Todd
G. Toennies
B. M. Tolbert
N. E. Tolbert
R. M. Tomarelli

G. M. Tomkins
Y. J. Topper
J. R. Totter
J. C. Touchstone
O. Touster
C. R. Treadwell
H. C. Trimble
W. Troll
R. E. Trucco
F. I. Tsuji
J. Tuba
R. A. Turner
A. A. Tytell
S. Udenfriend
H. E. Umbarger
W. W. Umbreit
L. A. Underkofler
M. F. Utter
B. L. Vallee
J. T. Van Bruggen
J. F. Van Pilsum
D. D. Van Slyke
H. Van Vunakis
W. J. van Wagtendonk
L. Varga
J. E. Varner
H. M. Vars
S. F. Velick
B. Vennesland
E. H. Venning
L. P. Vernon
C. S. Vestling
H. B. Vickery
C. A. Villee, Jr.
R. W. Virtue
W. Vishniac
D. W. Visser
H. J. Vogel
E. Volkin
R. W. Von Korff
J. Waddell
H. Waelsch
T. Wagner-Jauregg
W. W. Wainio
H. A. Waisman
S. J. Wakil
G. Wald
B. S. Walker
J. B. Walker
Z. Wallen-Lawrence
C. C. Wang
C. H. Wang
T.-y. Wang
A. G. Ware
R. C. Warner
R. E. Waterman
C. J. Watson
J. D. Watson
D. F. Waugh
C. J. Weber
G. C. Webster
T. E. Weichselbaum
L. Weil
E. C. Weinbach
S. Weinhouse
A. Weissbach
N. Weissman
B. Weissmann
A. D. Welch
I. C. Wells
W. B. Wendel
S. H. Wender
H. Werbin
C. H. Werkman
E. S. West
H. D. West
W. W. Westerfeld
F. H. Westheimer
U. F. Westphal
R. L. Whistler
A. White
A. G. C. White
F. R. White
J. White
W. F. White
H. R. Whiteley
A. N. Wick
W. G. Wiest
P. E. Wilcox
F. H. Wiley
A. E. Wilhelmi
V. A. Wilkerson
H. H. Williams
J. N. Williams, Jr.
J. W. Williams
R. J. Williams
V. R. Williams
W. L. Williams
H. G. Williams-Ashman
M. B. Williamson
D. W. Wilson
H. N. Wilson
I. B. Wilson
P. W. Wilson
T. H. Wilson
W. J. Wingo
M. Winitz
T. Winnick
O. Wintersteiner
R. J. Winzler
J. Wittenberg
R. F. Witter
G. Wolf
J. B. Wolfe
W. A. Wolff
M. L. Wolfrom
H. G. Wood
J. L. Wood
T. R. Wood
W. A. Wood
M. W. Woods
D. W. Woolley
W. D. Wosilait
H. H. Wotiz
B. E. Wright
L. D. Wright
S. L. Wright
H. Wu
G. R. Wyatt
A. M. Wynne
O. Wyss
C. Yanofsky
E. G. Young
I. Zabin
A. Zaffaroni
P. Zamecnik
S. Zamenhof
L. Zechmeister
I. Zelitch
E. A. Zeller
L. P. Zill
F. Zilliken
C. A. Zittle

RETIRED MEMBERS

W. H. Adolph
S. Amberg
W. E. Anderson
J. C. Andrews
C. V. Bailey
E. Baumann
C. Benson
O. Bergeim
N. R. Blatherwick
M. J. Blish
W. R. Bloor
J. C. Bock
A. Bodansky
L. J. Bogert
A. K. Boor
A. W. Bosworth
H. C. Bradley
A. P. Briggs
G. D. Buckner
H. H. Bunzell
W. R. Campbell
T. M. Carpenter
C. A. Cary
E. D. Clark
G. W. Clark
W. M. Clark
G. H. A. Clowes
D. H. Cook
H. J. Corper
L. H. Cretcher
A. L. Daniels
G. E. Delory
R. A. Dutcher
M. Dye
W. H. Eddy
A. A. Epstein
C. J. Farmer
H. O. L. Fischer
M. H. Fischer
C. H. Fiske
G. L. Foster
C. Funk
J. L. Gamble
E. M. K. Geiling
A. O. Gettler
R. B. Gibson
M. H. Givens
I. Greenwald
B. Harrow
E. N. Harvey
M. Heidelberger
V. G. Heller
J. S. Hepburn
F. F. Heyroth
A. M. Hjort
A. G. Hogan
P. E. Howe
R. B. Hubbell
A. Hunter
W. A. Jacobs
O. Kamm
E. C. Kendall
C. Kennedy
I. S. Kleiner
A. Knudson
P. A. Kober
R. A. Kocher
M. Kunitz
A. R. Lamb
R. C. Lewis
J. F. Lyman
A. B. Macallum
G. MacLeod
E. K. Marshall, Jr.
L. A. Maynard
W. S. McCann
J. F. McClendon
E. V. McCollum
F. H. McCrudden
H. A. McGuigan
J. S. McHargue
F. C. McLean
H. H. Mitchell
A. F. Morgan
S. Morgulis
J. Murlin
J. M. Nelson
H. T. Parsons
W. H. Peterson
E. J. Quinn
R. E. Remington
A. N. Richards
L. K. Riggs
C. S. Robinson
L. G. Rowntree
I. Sandiford
O. Schreiner
D. A. Scott
E. L. Scott
A. Seidell
C. P. Sherwin
T. Sollmann
M. Somogyi
W. C. Stadie
H. Steenbock
M. X. Sullivan
E. S. Sundstroem
B. Sure
R. E. Swain
S. Tashiro
C. Voegtlin
H. Wasteneys
L. G. Wesson
F. D. White
R. R. Williams
E. Woods

American Society for Parmacology and Experimental Therapeutics

ACTIVE MEMBERS

B. E. Abreu
G. H. Acheson
L. B. Achor
W. L. Adams
T. K. Adler
R. P. Ahlquist
M. Ajazzi-Mancini
G. A. Alles
A. M. Ambrose
C. M. Ambrus
J. L. Ambrus
H. H. Anderson
R. C. Anderson
H. V. Aposhian
J. F. Artusio, Jr.
D. M. Aviado, Jr.
J. Axelrod
N. Back
D. H. Baeder
J. A. Bain
W. W. Baker
S. B. Barker
O. W. Barlow
T. C. Barnes
A. D. Bass
W. B. Bass
R. C. Batterman
R. O. Bauer
J. H. Baxter
I. T. Beck
L. V. Beck
H. Beckman
H. K. Beecher
J. Belford
M. Belkin
J. W. Bellville
B. G. Benfey
J. M. Benforado

W. M. Benson
J. G. Benton
F. M. Berger
F. Bernheim
R. H. Beutner
K. H. Beyer, Jr.
H. R. Bierman
R. N. Bieter
W. P. Blackmore
M. R. Blair, Jr.
K. C. Blanchard
C. I. Bliss
A. L. Bloomfield
H. Blumberg
O. Bodansky
D. F. Bogdanski
W. P. Boger
D. D. Bonnycastle
W. M. Booker
H. L. Borison
W. Bourne
E. M. Boyd
E. S. Boyd
L. J. Boyd
E. D. Brand
A. C. Bratton, Jr.
R. W. Brauer
H. A. Braun
E. M. Bridge
H. Brieger
F. N. Briggs
B. B. Brodie
T. M. Brody
C. Brooks
C. McC. Brooks
B. B. Brown
R. V. Brown
H. D. Bruner
H. H. Bryant
A. M. Budy
E. Bueding
J. P. Bunker
T. N. Burbridge
A. S. V. Burgen
R. M. Burgison
J. C. Burke
J. H. Burn
J. J. Burns
H. Busch
M. T. Bush
T. C. Butler
E. J. Cafruny
R. L. Caben
B. Calesnick
N. O. Calloway
W. J. R. Camp
A. Cantarow
G. L. Cantoni
W. F. Cantrell
R. T. Capps
C. J. Carr
E. A. Carr, Jr.
McK. Cattell
D. J. Cavanaugh
R. J. Cazort
H. F. Chase
G. Chen
J. Y. Chen
K. K. Chen
R. H. Cheney
M. B. Chenoweth
G. P. Child
Y. C. Chin
H. I. Chinn
R. N. Chopra (Hon.)
B. B. Clark
W. G. Clark
D. A. Clarke
J. Cochin
K. W. Cochran
C. F. Code
V. V. Cole
R. J. Collins
J. H. Comroe, Jr.
G. M. Conzelman, Jr.
D. L. Cook
L. Cook
J. M. Coon
A. C. Corcoran
E. Costa
P. J. Costa
M. D. Cotten
G. K. Cotts
F. Co Tui
E. M. Cranston
B. N. Craver
P. J. Crittenden
G. E. Cronheim
A. P. Crosley, Jr.
C. W. Crumpton
T. Z. Csaky
S. C. Cullen
H. Cullumbine
R. W. Cunningham
W. C. Cutting
H. H. Dale (Hon.)
E. E. Daniel
T. D. Darby
N. A. David
B. Davidow
J. D. Davidson
B. D. Davis
J. E. Davis
M. E. Davis
O. F. Davis
C. Davison
N. N. De
E. H. Dearborn
E. J. de Beer
R. C. de Bodo
B. De Boer
F. DeEds
A. C. DeGraff
W. B. Deichmann
E. W. J. DeMaar
J. Dempsher
E. W. Dennis
W. E. Deturk
P. B. Dews
H. L. Dickison
H. F. Diermeier
J. M. Dille
J. B. Dillon
J. R. Di Palma
V. F. DiStefano
E. F. Domino
D. D. Donahue
A. S. Dontas
W. W. Douglas
J. Doull
C. A. Dragstedt
J. H. Draize
W. B. Draper
R. H. Dreisbach
P. E. Dresel
N. B. Dreyer
V. A. Drill
R. D. Dripps
R. L. Driver
K. P. Dubois
P. R. Dumke
G. S. Eadie
E. Eagle
N. B. Eddy
L. L. Eisenbrandt
J. J. Elkes
F. P. Ellinger
H. W. Elliott
F. W. Ellis
S. Ellis
G. A. Emerson
E. W. Emmart
N. Ercoli
D. W. Esplin
B. E. Etsten
G. M. Everett
G. Falk
A. E. Farah
E. B. Farnsworth
G. E. Farrar, Jr.
D. W. Fassett
A. Faulconer, Jr.
J. F. Fazekas
R. M. Featherstone
E. J. Fellows
F. C. Ferguson, Jr.
E. G. Fingl
J. K. Finnegan
O. G. Fitzhugh
F. F. Foldes
E. L. Foltz
R. B. Forney
R. H. K. Foster
J. G. Foulks
H. F. Fraser
J. P. Frawley
M. V. Freeman
E. D. Freis
W. A. Freyburger, Jr.
D. G. Friend
M. J. Frumin
N. W. Fugo
W. H. Funderburk
R. F. Furchgott
A. Furst
J. H. Gaddum
W. H. Gantt
A. Ganz
S. Garb
J. F. Gardocki
H. S. Gasser
E. M. K. Geiling
A. Gellhorn
C. L. Gemmill
R. George
S. B. Gertner
R. M. Gesler
N. J. Giarman
O. S. Gibbs
A. Gilman
H. G. Glass
J. M. Glassman
M. I. Gluckman
H. Gold
L. R. Goldbaum
L. I. Goldberg
A. Goldin
A. Goldstein
L. S. Goodman
R. E. Gosselin
A. Goth
D. R. H. Gourley
W. M. Govier
C. W. Gowdey
H. T. Graham
W. D. Graham
G. W. Gray
M. G. Gray
W. D. Gray
D. M. Green
H. D. Green
J. P. Green
R. E. Green
D. A. Greenwood
C. M. Greer
M. E. Greig
T. H. Greiner
K. S. Grimson
D. Grob
A. Grollman
E. G. Gross
C. M. Gruber, Jr.
C. C. Gruhzit
F. Guerra
L. Gyermek
H. B. Haag
Z. Hadidian
J. H. Hafkenschiel, Jr.
J. A. Hagans
P. B. Hagen
T. J. Haley
W. E. Hambourger
C. A. Handley
C. Hanna
E. L. Hansen
R. N. Harger
B. K. Harned
P. Harris
S. C. Harris
E. R. Hart
A. McG. Harvey
S. C. Harvey
N. Haugaard
W. J. Hayes, Jr.
J. M. Hayman, Jr.
H. W. Hays
L. W. Hazleton
M. A. Heinrich, Jr.
A. A. Hellbaum
A. E. Heming
L. C. Hendershot
F. G. Henderson
J. P. Hendrix
J. B. Herrmann
R. G. Herrmann
S. G. Hershey
R. P. Herwick
C. Heymans
J. Hidalgo
H. E. Hill
J. G. Hilton
H. E. Himwich
C. H. Hine
P. Hitchcock
H. C. Hodge
J. B. Hoekstra
F. G. Hofmann
C. A. M. Hogben
H. G. O. Holck
W. C. Holland
D. E. Holtkamp
J. O. Hoppe
A. Horita
R. W. Houde
K.-c. Huang
R. A. Huggins
H. R. Hulpieu
F. E. Hunter, Jr.
L. Hurwitz
D. E. Hutcheon
K. Hwang
D. D. Irish
S. Irwin
H. Isbell
R. M. Isenberger
H. K. Iwamoto
E. L. Jackson
E. Jacobsen
L. B. Jaques
M. E. Jarvik
D. J. Jenden
E. H. Jenney
G. Joachimoglu
L. M. Jones
R. Jones, Jr.
C. M. Kagawa
J. B. Kahn, Jr.
S. M. Kalman
W. Kalow
K. Kamijo
A. Kandel
A. G. Karczmar
L. Karel
D. A. Kamofsky
D. Karp
H. H. Keasling
A. S. Keats
F. E. Kelsey
F. K. O. Kelsey
C. J. Kensler
A. T. Kenyon
S. Kety
A. C. Keyl
E. K. Killam
K. F. Killam, Jr.
E. T. Kimura

K. K. Kimura
T. O. King
A. C. Kirchhof
P. K. Knoefel
Y. Kobayashi
J. J. Kocsis
G. B. Koelle
T. Koppanyi
C. Kornetsky
R. Koster
F. L. Kozelka
C. P. Kraatz
M. E. Krahl
J. C. Krantz, Jr.
O. Krayer
W. A. Krivoy
S. Krop
H. M. Krueger
H. A. Kuhn
E. Kun
H. S. Kupperman
N. T. Kwit
C. J. Lambertsen
A. M. Lands
H. Langemann
E. Larson
P. S. Larson
L. Lasagna
J. H. Last
E. P. Laug
W. S. Lawrence
A. H. Lawton
C. D. Leake
C.-C. Lee
H. M. Lee
K. S. Lee
A. J. Lehman
R. A. Lehman
D. Lehr
C. S. Leonard
R. R. Levine
R. L. Levy
J. R. Lewis
T. H. Li
P. A. Lief
E. W. Ligon, Jr.
R. K. S. Lim
C. R. Linegar
J. T. Litchfield
J. M. Little
E. R. Loew
L. Loewe
W. S. Loewe
O. Loewi (Hon.)
J. P. Long
T. A. Loomis
O. H. Lowry
F. C. Lu
G. Lu
M. Lubin
G. H. W. Lucas
F. P. Luduena
J. S. Lundy
F. C. MacIntosh
W. H. Macmillan
D. F. Magee
G. L. Maison
H. Maling
S. Mallov
H. G. Mandel
G. J. Mannering
T. E. Mansour
R. W. Manthei
T. H. Maren
S. Margolin
L. C. Mark
A. S. Marrazzi
E. K. Marshall, Jr.
F. M. Martin, Jr.
P. A. Martis
R. A. Maxwell
S. E. Mayer
E. A. Maynard
E. W. Maynert
W. P. McCann
E. L. McCawley
J. D. McColl
F. D. McCrea
A. R. McIntyre
H. McKennis, Jr.
G. R. McKinney
P. L. McLain
R. A. McLean
B. P. McNamara
M. K. McPhail
R. Meier
L. B. Mellett
K. I. Melville
C. Méndez
R. Mendez
F. H. Meyers
K. Mezey
E. Mihich
L. J. Milch
A. T. Milhorat
G. H. Miller
J. W. Miller
L. C. Miller
R. C. Millican
J. G. Millichap
L. C. Mills, Jr.
B. Minz
T. S. Miya
W. Modell
H. Molitor
J. Molland
N. C. Moran
C. F. Morgan
C. A. Morrell
L. E. Morris
P. E. Morrow
J. H. Moyer, III
G. H. Mudge
C. W. Muehlberger
B. Mukerji
M. G. Mulinos
P. L. Munson
J. E. Nadler
J. B. Nash
W. F. Neuman
I. Neuwirth
H. W. Newman
S.-h. Ngai
M. Nickerson
W. C. North
S. Norton
M. M. Nothman
F. W. Oberst
T. B. O'Dell
Y. T. Oester
G. T. Okita
J. F. O'Leary
E. Oppenheimer
J. A. Orcutt
L. R. Orkin
O. S. Orth
A. H. Owens, Jr.
R. G. Page
S. Y. P'An
E. M. Papper
E. G. Pardo
J. M. Parker
R. E. Parks, Jr.
L. G. Parmer
R. W. Payne
H. M. Peck
E. W. Pelikan
S. A. Peoples
P. L. Perlman
L. Peters
D. F. Petersen
C. C. Pfeiffer
N. M. Phatak
F. S. Philips
E. P. Pick
J. O. Pinkston
C. B. Pittinger
N. P. Plotnikoff
A. J. Plummer
S. N. Pradhan
J. B. Preston
H. L. Price
M. Prinzmetal
L. Procita
C. D. Proctor
A. J. Quick
J. P. Quigley
J. L. Radomski
D. P. Rall
L. O. Randall
L. A. Rantz
J. Reilly
J. F. Reinhard
B. R. Rennick
J. C. Rice
R. K. Richards
A. P. Richardson
J. A. Richardson
J. A. Rider
R. W. Ridley
F. Rieders
D. S. Riggs
W. F. Riker, Jr.
R. F. Riley
S. H. Rinzler
J. S. Robb
B. H. Robbins
J. Roberts
H. J. Robinson
M. Rocha e Silva
W. T. Rockhold
M. H. Roepke
R. R. Roepke
M. A. Root
H. Rosen
I. Rosenblum
M. Rosenfeld
S. M. Rosenthal
F. E. Roth
L. J. Roth
L. W. Roth
E. Rothlin
A. Rothstein
E. A. Rovenstine
B. Rubin
D. A. Rytand
J. Sacks
P. P. T. Sah
P. R. Salerno
L. T. Samuels
J. P. Saunders
P. R. Saunders
W. H. Sawyer
G. Sayers
W. B. Schallek
L. S. Schanker
C. F. Schmidt
J. L. Schmidt
L. H. Schmidt
J. A. Schneider
J. Scholler
M. Schubert
F. W. Schueler
F. H. Schultz, Jr.
C. C. Scott
K. G. Scott
J. V. Scudi
L. D. Seager
P. H. Seay
V. P. Seeberg
J. C. Seed
M. H. Seevers
R. A. Seibert
C. B. Shaffer
A. M. Shanes
J. A. Shannon
M. J. Shear
J. Seifter
T. Sherrod
F. E. Shideman
H. A. Shoemaker
P. A. Shore
C. O. Siebenmann
O. H. Siegmund
H. Silvette
A. Sjoerdsma
I. H. Slater
M. B. Slomka
A. E. Smith
C. C. Smith
C. M. Smith
D. J. Smith
D. L. Smith
P. K. Smith
P. W. Smith
R. B. Smith, Jr.
R. G. Smith
T. C. Smith
J. R. Smythies
T. Sollmann
J. N. Spencer
F. Sperling
T. D. Spies
M. A. Spirtes
J. N. Stannard
I. Starr
G. W. Stavraky
F. Steigmann
J. E. Steinhaus
E. F. Stohlman
H. E. Stokinger
A. Stoll
C. A. Stone
R. T. Stormont
J. L. Strominger
H. C. Struck
F. M. Sturtevant
J. W. Stutzman
C.-Y. Sung
E. W. Sutherland, Jr.
V. C. Sutherland
J. L. Swirbely
H. H. Swain
E. E. Swanson
E. A. Swinyard
I. I. A. Tabachnick
C. W. Tabor
H. Tabor
M. L. Tainter
D. B. Taylor
J. D. Taylor
R. S. Teague
D. H. Tedeschi
J. Tepperman
S. Thesleff
C. H. Thienes
J. E. Thomas
M. R. Thompson
P. S. Timiras
R. Tislow
E. O. Titus
J. E. P. Toman
J. H. Trapold
J. Travell
V. G. Trendelenburg
A. P. Truant
E. B. Truitt, Jr.
D. B. Tyler
S. Udenfriend
K. R. W. Unna
R. C. Ursillo
J. R. Valle
C. G. Van Arman
M. J. Vander Brook
H. B. van Dyke
E. F. Van Maanen
W. Van Winkle, Jr.
V. G. Vernier
R. L. Vick
W. F. Von Oettingen
B. J. Vos
W. J. Waddell
G. E. Wakerlin
K. G. Wakim
R. P. Walton
R. I. H. Wang

S. C. Wang
C. P. Wangeman
M. R. Warren
D. T. Watts
R. A. Waud
E. L. Way
J. L. Way
J. H. Weatherby
L. C. Weaver
R. Wegria
J. H. Weikel, Jr.
A. D. Welch
J. A. Wells
H. Wendel
G. Werner
H. W. Werner
W. C. Wescoe
L. G. Wesson
T. C. West
B. A. Westfall
C. W. White, Jr.
P. D. White
R. W. Whitehead
P. Whittlesey
A. Wikler
W. Wilbrandt
J. H. Wills
R. H. Wilson
M. M. Winbury
C. V. Winder
C. A. Winter
I. C. Winter
W. D. Winters
P. N. Witt
H. G. Wolff
A. Wollenberger
D. R. Wood
E. H. Wood
G. Woodard
D. M. Woodbury
R. A. Woodbury
E. F. Woods
L. A. Woods
D. W. Woolley
C. I. Wright
H. N. Wright
F. F. Yonkman
W. B. Youmans
A. G. Young
T.-f. Yu
V. Zapata-Ortiz
H. L. Zauder
C. G. Zubrod

RETIRED MEMBERS

W. C. Alvarez
S. Amberg
A. Arkin
E. D. Brown
M. S. Dooley
E. F. Du Bois
H. Eagle
H. E. Essex
J. A. E. Eyster
G. Fahr
G. P. Grabfield
C. M. Gruber
C. C. Guthrie
W. Hale
D. E. Jackson
W. A. Jacobs
N. M. Keith
W. J. Kerr
I. S. Kleiner
T. K. Kruse
P. D. Lamson
A. E. Livingston
D. I. Macht
H. A. McGuigan
F. C. McLean
E. E. Nelson
I. H. Pierce
B. O. Raulston
A. N. Richards
J. M. Rogoff
G. B. Roth
L. G. Rowntree
C. Voegtlin
R. M. Waters
C. J. Wiggers

American Society for Experimental Pathology

ACTIVE MEMBERS

J. M. Adams
F. N. Allan
F. M. Allen
E. L. Alling
C. H. Alrshuler
E. C. Alvord, Jr.
D. H. Andersen
W. A. D. Anderson
H. B. Andervont
R. H. Andresen
S. B. Andrus
D. M. Angevine
W. Antopol
F. L. Apperly
J. S. Arnold
L. Arnold
J. D. Aronson
C. A. Ashley
S. H. Ashbach
O. T. Bailey
A. B. Baker
J. A. Baker
R. D. Baker
H. A. Ball
F. B. Bang
W. A. Bardawil
J. H. Bauer
P. R. Beamer
J. W. Beard
C. S. Beck
W. J. Beckfield
D. L. Belding
E. T. Bell
S. A. Bencosme
E. P. Benditt
K. Benirschke
G. A. Bennett
L. R. Bennett
W. A. Bennett
B. N. Berg
F. H. Bethell
M. Bevans
G. R. Biskind
D. H. Blankenhorn
M. H. Block
J. M. G. Bloodworth, Jr.
W. Bloom
A. L. Bloomfield
H. T. Blumenthal
J. W. Blum
C. G. Bly
J. L. Bollman
W. L. Bostwick
P. E. Boyle
J. H. Bragdon
E. A. G. Branch
W. W. Brandes
H. Braunstein
G. Brecher
C. Breedis
K. M. Brinkhous
G. O. Broun
A. L. Brown, Jr.
A. Brunschwig
J. G. Brunson
W. R. Bryan
G. J. Buddingh
J. C. Bugher
E. L. Burns
M. S. Burstone
P. R. Cannon
W. H. Carnes
J. L. Carr
G. M. Carrera
J. R. Carter
G. W. Casarett
A. E. Casey
J. R. Cash
W. B. Castle
B. Castleman
L. Catron
G. W. Changus
A. M. Chesney
J. J. Christian
R. W. Christie
J. J. Clemmons
J. Cohen
D. R. Coman
C. C. Congdon
R. W. Coon
D. R. Cordy
G. C. Cozzias
D. Cowen
A. J. Cox, Jr.
J. M. Craig
W. J. Cromartie
L. V. Crowley
L. K. Dahl
G. Dalldorf
A. J. Dalton
I. Davidsohn
H. A. Davis
R. LaV. Davis
J. R. Dawson, Jr.
C. G. de Gutierrez-Mahoney
J. de la Huerga
E. de Savitsch
F. J. Dixon, Jr.
H. M. Dixon
C. A. Doan
I. N. Dubin
C. E. Dunlap
T. B. Dunn
W. R. Earle
E. E. Ecker
E. A. Eckert
J. E. Edwards
R. H. Egdahl
A. H. Eggerth
W. Ehrich
E. J. Eichwald
C. M. Eklund
J. T. Ellis
K. M. Endicott
C. C. Erickson
A. B. Eschenbrenner
V. J. Evans
E. Farber
S. Farber
L. E. Farr
R. S. Farr
J. D. Feldman
W. H. Feldman
L. K. Ferguson
R. L. Ferguson
M. P. Finkel
H. I. Firminger
E. R. Fisher
F. W. Fitch
P. J. Fitzgerald
M. S. Fleisher
J. E. Flynn
J. H. Fodden
L. C. Fogg
G. E. Foley
R. H. Follis, Jr.
A. G. Foraker
B. K. Forscher
T. Francis, Jr.
D. G. Freiman
J. K. Frenkel
G. H. Fried
W. F. Friedewald
N. B. Friedman
J. Furth
E. A. Gall
O. Gaes
J. R. Geary, Jr.
Q. Geiman
F. G. Germuth
I. Gersh
R. J. Glaser
C. A. Gleiser
J. W. Goddard
J. T. Godwin
H. Goldblatt
A. Golden
R. A. Good
E. W. Goodpasture
F. B. Gordon
J. R. Gorham
H. G. Grady
L. Graef
J. B. Graham
R. C. Grauer
C. H. Greene
H. S. N. Greene
P. Gross
R. E. Gross
O. M. Gruhzit
P. M. Gullino
F. D. Gunn
R. T. Habermann
D. B. Hackel
P. F. Hahn
W. M. Hale
B. Halpert
J. A. Halsted
A. W. Ham
J. D. Hamilton
L. D. Hamilton
T. R. Hamilton
W. McD. Hammon
C. W. Hammond
A. H. Handler
A. F. Hansen
J. C. Harkin
J. W. Harman
W. J. Harrington
C. Harris
P. Harris
W. H. Harris
F. W. Hartman
W. S. Hartroft
G. Haas
W. B. Hawkins
C. V. Z. Hawn
E. E. Hellerstein
R. C. Hendrix
G. R. Hennigar

O. Hepler
L. G. Herrmann
A. Hertig
W. E. Heston
S. P. Hicks
B. Highman
W. T. Hill
J. R. Hinshaw
C. Hoch-Ligeti
F. W. Hoffbauer
R. L. Holman
F. Homburger
H. C. Hoppe
J. W. Howland
W. C. Hueper
D. M. Hume
D. T. Imagawa
J. R. M. Innes
R. Isaacs
L. O. Jacobson
H. L. Jaffe
W. E. Jaques
F. L. Jennings
R. B. Jennings
W. Joel
E. E. Jones
R. S. Jones
C. W. Jungeblut
R. L. Kahn
H. S. Kaplan
N. Kaufman
H. Kaunitz
N. M. Keith
A. Kellner
S. P. Kent
H. D. Kesten
J. G. Kidd
T. D. Kinney
R. Kinosita
A. Kirschbaum
M. Klein
J. Kleinerman
S. Koletsky
R. Korson
P. Kotin
C. A. Krakower
A. P. Krueger
L. S. Kubie
J. P. Kulka
M. Kuschner
P. E. Lacy
A. K. Laird
J. J. Lalich
R. A. Lambert
B. G. Lamson
B. H. Landing
R. D. Langdell
K. Lange
G. L. Laqueur
H. Larra
L. W. Law
S. S. Lazarus
E. H. Leduc
J. Leighton
E. H. Lennette
E. M. Lerner, II
C. Leuchtenberger
S. A. Levinson
B. M. Levy
J. H. Lewis
R. D. Lillie
S. Lindsay
S. W. Lippincott
H. Lisco
L. Loeb
G. A. Lo Grippo
L. S. Lombard
W. E. Loring
R. Love
C. U. Lowe
F. V. Lucas
G. D. Lumb
M. B. Lurie
S. A. Luse
C. C. Lushbaugh
R. C. MacCardle
S. C. Madden
C. L. Maddock
S. Maddock
H. J. Magnuson
G. Majno
G. K. Mallory
R. A. Malmgren
F. D. Mann
J. D. Mann
W. Manwaring
G. Margolis
E. J. Mason
M. McCutcheon
H. C. McGill, Jr.
D. G. McKay
F. W. McKee
J. F. A. McManus
P. D. McMaster
J. B. McNaught
I. McQuarrie
W. A. Meissner
R. C. Mellors
G. R. Meneely
V. Menkin
M. L. Menten
S. R. Mettler
J. B. Miale
G. B. Mider
C. P. Miller
F. R. Miller
R. W. Monto
H. D. Moon
C. V. Moore
R. A. Moore
R. D. Moore
T. J. Moran
R. H. More
A. R. Moritz
S. Mudd
E. E. Muirhead
R. M. Mulligan
G. E. Murphy
C. W. Mushett
E. M. Nadel
A. A. Nelson
E. L. Nelson
J. B. Nelson
A. Nettleship
J. M. Nichols
E. T. Nishimura
T. D. Norman
J. R. Oliver
C. Olson, Jr.
R. M. O'Neal
J. L. Orbison
L. G. Ortega
P. Ortega
C. A. Owen, Jr.
T. C. Panos
E. A. Park
R. F. Parker
L. W. Parr
R. J. Parsons
J. C. Paterson
J. M. Pearce
H. E. Pearse
B. Pearson
G. D. Penick
H. H. Permar
Van B. Philpot, Jr.
C. F. Piel
H. Pinkerton
C. L. Pirani
A. D. Pollack
O. J. Pollak
M. Pollard
H. Popper
R. T. Prehn
H. C. Pribor
F. B. Queen
A. S. Rabson
C. C. Randall
H. L. Ratcliffe
L. J. Rather
L. S. Ravdin
J. W. Rebuck
J. D. Reese
H. A. Reimann
S. P. Reimann
P. E. Rekers
C. P. Rhoads
A. R. Rich
G. W. Richter
M. N. Richter
R. H. Rigdon
T. M. Rivers
S. L. Robbins
F. S. Robscheit-Robbins
S. Rogers
L. Roizin
P. D. Rosahn
S. R. Rosenthal
J. F. Ross
O. A. Ross
P. Rous
D. A. Rowley
H. P. Rusch
W. O. Russell
A. B. Russfield
J. H. Rust
K. K. Sanford
O. Saphir
D. G. Scarpelli
F. M. Schabel, Jr.
H. J. Schattenberg
J. R. Schenken
G. W. H. Schepers
J. A. Schilling
H. G. Schlumberger
H. C. Schmeisser
R. Schmid
R. Schrek
E. W. Schultz
J. K. Scott
D. P. Seecof
B. C. Seegal
A. Segaloff
M. F. Shaffer
J. N. Shanberge
J. L. Shapiro
E. Shelton
M. B. Shimkin
R. E. Shope
P. Shubik
G. Shwartzman
W. J. Siebert
M. R. Sikov
M. Silberberg
R. Silberberg
J. P. Simonds
I. Singer
F. R. Skelton
J. E. Smadel
J. C. Small
H. F. Smetana
A. G. Smith
H. P. Smith
L. W. Smith
R. T. Smith
A. M. Snell
L. Sokoloff
S. C. Sommers
B. H. Spargo
T. D. Spies
E. E. Sproul
D. H. Sprunt
F. W. Stamler
M. F. Stanton
R. B. Stebbins
M. Stefanini
C. H. Steffee
B. Steinberg
P. E. Steiner
R. C. Stepto
K. Stern
C. A. Stetson
F. W. Stewart
H. L. Stewart
W. B. Stewart
H. C. Stoerk
F. Stohlman
R. D. Stoner
J. B. Storer
R. E. Stowell
J. P. Strong
C. C. Sturgis
J. T. Syverton
E. B. Taft
A. Tannenbaum
C. B. Taylor
H. E. Taylor
H. M. Taylor
K. L. Terplan
R. Terry
A. P. Thal
W. A. Thomas
W. T. S. Thorp
W. J. Tomlinson
E. H. Tompkins
V. N. Tompkins
H. W. Toolan
F. M. Townsend
W. Trager
J. L. Tullis
A. C. Upton
J. L. Van Lancker
G. F. Vawter
J. J. Vazquez
R. L. Vernier
F. S. Vogel
B. W. Volk
E. von Haam
A. J. Vorwald
M. Wachstein
B. M. Wagner
R. L. Walford
C. W. Walter
O. H. Wangensteen
E. D. Warner
S. Warren
W. B. Wartman
L. W. Wattenberg
A. Weil
C. Weiss
A. A. Werder
J. K. Weston
B. C. Wexler
S. L. Wilens
G. Z. Williams
R. B. Williams, Jr.
R. H. Williams
W. Lane Williams
William L. Williams
J. W. Wilson
M. M. Wintrobe
B. K. Wiseman
R. W. Wissler
A. Wolf
E. Woll
C. R. Womack
S. Wood, Jr.
A. Wright
A. W. Wright
J. M. Young
C. L. Yuile
N. Zamcheck
I. Zeidman
L. J. Zeldis
B. Zimmerman
H. M. Zimmerman

RETIRED MEMBERS

S. Bayne-Jones
C. H. Bunting
R. Cole
G. M. Curtis
A. R. Dochez
C. W. Duval

N. C. Foot
G. R. Herrmann
J. W. Jobling
H. T. Karsner
E. C. Kendall
E. B. Krumbhaar
G. R. Lacy
E. R. Long
C. G. Loosli
F. C. Mann
D. Marine
K. F. Meyer
V. H. Moon
J. J. Morton
J. P. O'Hare
W. W. Oliver
E. L. Opie
J. R. Paul
L. Pearce
O. H. Robertson
E. C. Rosenow
C. Ten Broeck
G. H. Whipple
R. M. Wilder
M. C. Winternitz
I. T. Zeckwer

American Institute of Nutrition

ACTIVE MEMBERS

E. Aaes-Jorgensen
G. Adams
W. H. Adolph
R. B. Alfin-Slater
J. B. Allison
H. J. Almquist
J. T. Anderson
W. E. Anderson
A. Arnold
L. Arnrich
G. Arroyave
C. F. Asenjo
L. Atkin
J. Aub
A. E. Axelrod
J. J. Barboriak
R. H. Barnes
E. L. Batchelder
J. C. Bauernfeind
C. A. Baumann
L. A. Bavetta
E. F. Beach
D. E. Becker
W. M. Beeson
O. G. Bentley
C. P. Berg
F. W. Bernhart
O. A. Bessey
R. M. Bethke
J. M. R. Beveridge
J. G. Bieri
C. E. Bills
F. G. Bing
F. H. Bird
H. R. Bird
F. E. Bischoff
A. Black
R. J. Block
H. Blumberg
L. E. Booher
R. Borchers
D. K. Bosshardt
R. V. Boucher
H. D. Branion
J. W. Bratzler
R. Bressani
W. D. Brewer
E. B. Bridgforth
G. M. Briggs
M. Brin
M. C. H. Brookes
J. B. Brown
R. A. Brown
J. Brozek
H. B. Burch
J. H. Burchenal
B. S. Burke
G. O. Burr
W. Burroughs
L. C. Butler
H. R. Butt
J. S. Butts
F. A. Cajori
M. L. Caldwell
D. H. Calloway
J. A. Campbell
C. W. Carrick
H. E. Carter
G. E. Cartwright
D. Catron
D. C. Cederquist
L. R. Cerecedo
W. H. Chambers
J. P. Chandler
M. W. Chang
C. Chatfield
V. H. Cheldelin
A. L. S. Cheng
B. F. Chow
M. F. Clarke
C. L. Comar
G. F. Combs
C. M. Coons
J. M. Cooperman
J. R. Couch
G. R. Cowgill
G. J. Cox
W. M. Cox, Jr.
E. W. Crampton
W. W. Cravens
T. J. Cunha
E. Da Costa
F. S. Daft
R. G. Daggs
H. Dam
L. J. Daniel
W. J. Darby
W. Dauler
B. L. Davey
C. S. Davidson
G. K. Davis
H. G. Day
P. L. Day
C. A. Denton
J. S. Dinning
M. L. Dodds
D. L. Drabkin
T. G. H. Drake
L. P. Dryden
V. du Vigneaud
H. M. Dyer
H. D. Eaton
M. M. Elliot
L. N. Ellis
N. R. Ellis
C. A. Elvehjem
G. A. Emerson
R. W. Engel
E. S. Eppright
B. H. Ershoff
R. J. Evans
G. Everson
P. F. Fenton
M. L. Fincke
H. Fisher
K. H. Fisher
K. Folkers
R. M. Forbes
M. R. S. Fox
G. S. Fraenkel
A. H. Free
C. E. French
C. N. Frey
T. E. Friedemann
L. Friedman
J. C. Fritz
D. V. Frost
C. Funk
E. Geiger
S. N. Gershoff
R. P. Geyer
M. B. Gillis
M. Goettsch
G. A. Goldsmith
H. H. Gordon
R. A. Gortner, Jr.
H. Goss
C. E. Graham
F. Grande
C. R. Grau
S. M. Greenberg
W. H. Griffith
A. C. Groschke
C. J. Gubler
N. B. Guerrant
P. György
J. R. Haag
J. E. Halver
T. S. Hamilton
P. Handler
S. L. Hansard
A. F. Hansen
A. E. Harper
L. E. Harris
P. L. Harris
R. S. Harris
R. A. Harte
A. M. Hartman
E. C. Hartman
A. B. Hastings
M. L. Hathaway
H. M. Hauck
E. E. Hawley
D. M. Hegsted
R. W. Heinle
L. M. Henderson
J. H. Hewetson
E. M. Hewston
B. W. Heywang
J. W. Hibbs
E. G. High
C. H. Hill
F. W. Hill
F. A. Hitchcock
J. A. Hoefer
A. G. Hogan
R. T. Holman
J. O. Holmes
L. E. Holt, Jr.
I. M. Hoobler
C. A. Hoppert
M. K. Horwitt
H. C. Hou
E. L. Hove
C. F. Huffman
J. M. Hundley
H. A. Hunscher
J. E. Hunter
A. C. Ivy
R. L. Jackson
R. W. Jackson
B. C. Johnson
P. E. Johnson
R. E. Johnson
S. R. Johnson
F. A. Johnston
N. Jolliffe
J. H. Jones
T. H. Jukes
B. M. Kagan
J. Kastelic
E. O. Keiles
T. B. Keith
A. R. Kemmerer
J. C. Keresztesy
M. C. Kik
C. G. King
L. W. Kinsell
M. Kleiber
S. Kletzien
O. L. Kline
C. B. Knodt
A. Knudson
C. J. Koehn
M. Kramer
F. H. Kratzer
W. E. Krauss
H. F. Kraybill
W. A. Krehl
C. H. Krieger
H. D. Kruse
F. A. Kummerow
D. M. Kydd
A. R. Lamb
E. J. Lease
M. O. Lee
A. L. Lehninger
J. M. Leichsenring
S. Lepkovsky
R. M. Leverton
S. Levey
H. Levine
S. Z. Levine
G. K. Lewis
I. E. Liener
R. J. Lillie
L. E. Lloyd
H. E. Longenecker
J. K. Loosli
T. D. Luckey
R. W. Luecke
C. M. Lyman
L. J. Machlin
C. G. Mackenzie
F. L. MacLeod
R. MacVicar
L. L. Madsen
G. V. Mann
M. E. Marsh
K. E. Mason
G. Matrone
L. D. Matterson
F. H. Mattson
C. D. May
J. Mayer
L. A. Maynard
W. S. McCann
C. M. McCay
F. J. McClure
E. B. McCollum
E. V. McCollum
R. H. McCoy
F. G. McDonald
L. W. McElroy

W. J. McGanity
J. McGinnis
E. W. McHenry
B. A. McLaren
J. Meites
D. Melnick
E. T. Mertz
F. L. Meyer
O. Mickelsen
A. T. Milhorat
C. D. Miller
O. N. Miller
R. C. Miller
R. F. Miller
H. H. Mitchell
H. S. Mitchell
C. V. Moore
L. A. Moore
A. F. Morgan
H. P. Morris
A. B. Morrison
E. Z. Moyer
H. E. Munsell
T. K. Murray
E. S. Nasset
E. M. Nelson
M. M. Nelson
W. L. Nelson
H. M. Nitowsky
L. C. Norris
B. L. O'Dell
L. M. Odland
M. A. Ohlson
R. Okey
H. Oldham
R. E. Olson
A. U. Orten
J. M. Orten
B. L. Oser
W. H. Ott
D. B. Parrish
H. Patrick
M. B. Patton
P. B. Pearson
W. N. Pearson
D. W. Peterson
L. B. Pett
W. H. Pfander
J. J. Pfiffner
P. H. Phillips
H. B. Pierce
R. L. Pike
H. Pollack
T. Porter
F. W. Quackenbush
M. L. Qualfe
E. P. Ralli
M. E. Reid
E. H. Reisner, Jr.
M. S. Reynolds
E. E. Rice
L. R. Richardson
L. J. Roberts
E. C. Robertson
H. E. Robinson
C. E. Roderuck
J. H. Roe
P. K. Roine
H. F. Root
W. C. Rose
F. Rosen
H. R. Rosenberg
J. I. Rowntree
S. H. Rubin
L. L. Rusoff
W. D. Salmon
M. Sampson
L. T. Samuels
M. R. Sandels
F. O. Santos
H. P. Sarett
P. S. Sarma
G. H. Satterfield
H. E. Sauberlich
A. E. Schaefer
F. H. Scharles
B. H. Schneider
H. A. Schneider
C. Schuck
M. O. Schultze
K. Schwarz
B. S. Schweigert
H. M. Scott
M. L. Scott
F. I. Scoular
N. S. Scrimshaw
J. V. Scudi
W. H. Sebrell, Jr.
E. L. Severinghaus
G. R. Sharpless
J. H. Shaw
W. C. Sherman
M. E. Shils
R. L. Shirley
H. Sinclair
S. A. Singal
A. H. Smith
D. T. Smith
J. M. Smith
S. C. Smith
S. E. Smith
E. E. Snell
S. E. Snyderman
H. H. Sobotka
S. Soskin
H. Spector
M. Speirs
T. D. Spies
A. Spohn
R. L. Squibb
F. J. Stare
G. Stearns
B. F. Steele
H. Steenbock
J. A. Stekol
E. L. Stephenson
H. K. Stiebeling
E. L. R. Stokstad
C. A. Storvick
M. L. Sunde
T. S. Sutton
P. Swanson
R. W. Swift
V. P. Sydenstricker
C. M. Taylor
M. W. Taylor
A. E. Teeri
L. J. Teply
B. H. Thomas
H. C. Tidwell
H. W. Titus
W. R. Todd
E. N. Todhunter
C. D. Tolle
R. M. Tomarelli
D. Tourtellotte
C. R. Treadwell
M. F. Trulson
R. G. Tucker
W. G. Unglaub
K. R. W. Unna
E. M. Vahlteich
T. B. Van Itallie
R. Van Reen
H. M. Vars
J. J. Vitale
L. Voris
J. Waddell
H. D. Wallace
C. C. Wang
R. H. Wasserman
D. M. Watkin
B. Webster
A. W. Wertz
N. C. Wetzel
A. White
P. L. White
C. K. Whitehair
A. C. Wiese
H. F. Wiese
H. H. Williams
R. D. Williams
R. R. Williams
E. D. Wilson
G. H. Wise
R. L. Wixom
M. G. Wohl
M. Womack
C. W. Woodruff
D. W. Woolley
L. D. Wright
J. B. Youmans
C. M. Young
R. J. Young
I. Zipkin

RETIRED MEMBERS

L. Ascham
C. V. Bailey
H. G. Barott
B. Bisbey
N. R. Blatherwick
A. W. Bosworth
H. L. Campbell
T. M. Carpenter
A. L. Daniels
E. F. Dubois
R. A. Dutcher
M. Dye
W. H. Eddy
E. B. Forbes
J. L. Gamble
I. Greenwald
F. R. Griffith, Jr.
A. Gulick
V. G. Heller
G. M. Higgins
H. L. Higgins
P. E. Howe
R. B. Hubbell
J. S. Hughes
E. P. Joslin
C. Kennedy
I. S. Kleiner
M. Koehne
R. C. Lewis
J. F. Lyman
G. MacLeod
J. F. McClendon
I. McQuarrie
J. Marlin
P. M. Nelson
H. T. Parsons
W. H. Peterson
M. S. Pittman
E. J. Quinn
R. E. Remington
I. Sandiford
S. L. Smith
B. Sure
J. Tilt
H. C. Trimble
R. M. Wilder
E. Woods

American Association of Immunologists

ACTIVE MEMBERS

W. W. Ackermann
F. L. Adler
F. Aladjem
P. M. Aptekman
L. Arnold
J. D. Aronson
E. R. Arquilla
W. M. Ashby
A. E. Axelrod
H. Baer
E. E. Baker
F. B. Bang
L. A. Barnes
H. C. Batson
J. W. Beard
E. R. Becker
S. M. Beiser
B. Benacerraf
A. A. Benedict
A. S. Benenson
O. R. Benham
I. L. Bennett, Jr.
T. O. Berge
D. T. Berman
A. W. Bernheimer
G. P. Berry
O. Bier
F. L. Black
E. A. Bliss
E. H. Bloch
H. Bloch
D. Bodian
G. C. Bond
A. Bondi
D. A. Boroff
K. L. Bowman
W. C. Boyd
A. A. Boyden
J. Bozicevich
J. H. Brewer
B. A. Briody
C. P. Brown
E. A. Brown
G. C. Brown
M. H. Brown
R. Brown
W. R. Bryan
E. L. Buescher
S. C. Bukantz
W. E. Bunney
K. L. Burdon
W. Burrows
A. Bussard
J. Caminopetros
D. H. Campbell
J. R. Cann
P. R. Cannon
W. A. Cassel
P. A. Cavelti
L. A. Chambers
M. W. Chase
H. N. Chatterjee
F. S. Cheever
B. F. Chow
L. R. Christensen
L. E. Cluff

A. F. Coburn
A. F. Coca (Hon.)
K. W. Cochran
J. M. Coffey
S. M. Cohen
R. A. Cooke
A. H. Coons
M. L. Cooper
E. J. Coulson
H. R. Cox
L. H. Criep
E. C. Curnen
J. E. Cushing
G. Dalldorf
J. B. Daniels
F. M. Davenport
I. Davidsohn
D. J. Davis
C. J. DeBoer
P. F. de Gara
E. W. Dennis
F. W. Denny, Jr.
H. A. Dettwiler
L. Dienes
J. H. Dingle
F. J. Dixon, Jr.
M. Dodd
C. E. Dolman
D. M. Donaldson
C. M. Downs
R. Drew
R. J. Dubos
C. E. Duffy
A. D. Dulaney
W. B. Dunham
M. Dworetzky
H. Eagle
M. D. Eaton
E. E. Ecker
E. A. Eckert
B. E. Eddy
G. Edsall
H. N. Eisen
S. S. Elberg
S. A. Ellison
J. F. Enders
J. B. Enright
J. O. Erickson
C. A. Evans
E. E. Evans
C. R. Falk
R. S. Farr
R. J. Feinberg
S. M. Feinberg
H. A. Feldman
A. E. Feller
O. Felsenfeld
L. C. Ferguson
M. A. Fink
M. Finland
E. E. Fischel
M. S. Fleisher
J. A. Flick
A. L. Florman
C. Floyd
E. M. Follensby
J. Fong
S. B. Formal
LeR. D. Fothergill
C. L. Fox, Jr.
J. P. Fox
T. Francis, Jr.
V. Fredette
R. Freter
J. Freund
C. Friend
H. S. Fuller
J. D. Fulton
W. L. Gaby
S. Gaines
A. L. Garbat
J. S. Garvey
L. N. Gay
L. P. Gebhardt
N. Gengozian
E. B. Gerheim
F. G. Germuth
H. M. Gezon
J. Gibbard
H. S. Ginsberg
D. Gitlin
W. S. Gochenour, Jr.
W. F. Goebel
H. Goldie
O. J. Golub
R. A. Good
M. Goodman
K. Goodner
I. Gordon
J. R. Gorham
R. Y. Gottshall
P. Grabar
R. H. Green
L. Greenberg
J. E. Gregory
A. Griffin
V. Groupe
K. Habel
S. Haberman
S. P. Halbert
W. M. Hale
W. McD. Hammon
D. Hamre
F. M. Hanger
J. H. Hanks
C. G. Harford
A. H. Harris
T. N. Harris
J. A. Harrison
R. W. Harrison
F. A. Haurowitz
S. P. Hayes
E. L. Hazen
M. Heidelberger
J. L. Hendry
W. Henle
A. V. Hennessy
M. Hilleman
G. K. Hirst
G. L. Hobby
A. B. Hoerlein
H. C. Hopps
H. E. B. Hopps
F. L. Horsfall, Jr.
D. M. Horstmann
G. A. Hottle
C. Howe
H. A. Howe
G. D. Hsiung
R. J. Huebner
T. P. Hughes
K. Hummeler
J. C. Humphries
M. S. Ingalls
J. Ipsen, Jr.
M. R. Irwin
K. Ishizaka
E. B. Jackson
L. Jacobs
B. V. Jager
E. Jawetz
R. K. Jennings
K. E. Jensen
W. S. Jeter
A. G. Johnson
W. S. Jordan
C. W. Jungeblut
E. A. Kabat
I. G. Kagan
R. L. Kahn
S. S. Kalter
A. S. Kaplan
M. H. Kaplan
F. Karush
D. T. Karzon
E. H. Kass
C. H. Kempe
F. E. Kendall
J. F. Kent
H. B. Kenzon
J. G. Kidd
E. D. Kilbourne
L. Kilham
S. F. Kitchen
W. L. Koerber
H. Koprowski
L. Korngold
M. E. Koshland
L. O. Krampitz
A. P. Krueger
W. J. Kuhns
D. B. Lackman
J. G. Lacorte
R. C. Lancefield
M. Landy
F. Lanni
C. L. Larson
D. L. Larson
H. S. Lawrence
E. H. Lennette
M. Leon
C. A. Leone
I. H. Lepow
S. Leskowitz
E. S. L'Esperance
L. Levine
P. Levine
H. L. Ley, Jr.
C. P. Li
S. J. Liso
R. L. Libby
R. B. Lindberg
M. M. Lipton
C. Liu
O. C. Liu
I. Live
G. A. Lo Grippo
C. G. Loosli
M. H. Loveless
F. C. Lowell
H. Lubinski
S. E. Luria
M. B. Lurie
W. N. Mack
C. M. MacLeod
T. P. Magill
T. Makinodan
S. Malkiel
G. P. Manire
W. Manwaring
S. Marcus
J. Marmorston
D. S. Martin
S. P. Martin
H. C. Mason
R. P. Mason
S. P. Masouredis
D. R. Mathieson
G. A. Matson
F. D. Maurer
P. H. Maurer
M. M. Mayer
M. McCarty
M. E. McClain
L. S. McClung
A. P. McKee
C. M. McKee
I. W. McLean, Jr.
W. F. McLimans
P. D. McMaster
C. McNeil
G. Meiklejohn
L. R. Melcher
R. C. Mellors
J. L. Melnick
V. Menkin
K. F. Meyer
C. P. Miller
R. E. Miller
G. S. Mirick
H. H. Mitchell
J. F. Mohn
N. Molomut
A. E. Moore
C. Morgan
H. R. Morgan
J. A. Morris
M. C. Morris
M. Moskowitz
I. M. Mountain
A. W. Moyer
S. Mudd
R. H. Muller
J. J. Munoz
E. G. D. Murray
F. J. Murray
R. Murray
L. H. Muschel
Q. N. Myrvik
J. M. Neill
C. T. Nelson
E. Neter
H. Neurath
W. J. Nungester
T. Ogata
B. J. Olson
A. G. Osler
J. Oudin
Z. Ovary
J. R. Overman
R. D. Owen
A. Packchanian
C. F. Pait
J. W. Palmer
A. M. Pappenheimer, Jr.
I. A. Parfentjev
R. F. Parker
P. Y. Paterson
J. R. Paul
H. E. Pearson
E. Perlman
M. Pittman
O. J. Plescia
J. R. Porter
H. M. Powell
D. Pressman
T. T. Puck
J. J. Quilligan, Jr.
F. M. Rackemann
S. Raffel
C. H. Rammelkamp
C. C. Randall
L. Rane
I. Rappaport
A. F. Rasmussen
M. Raynaud
J. F. Reback
M. Reissig
C. E. Rice
T. M. Rivers
F. C. Robbins
K. C. Robbins
M. L. Robbins
E. S. Robinson
H. M. Rose
N. R. Rose
T. Rosebury
I. Ruchman
R. Rustigian
A. B. Sabin
J. E. Salk
S. B. Salvin
S. Saslaw
M. Scherago
W. F. Scherer
H. W. Scherp
B. Schick
R. W. Schlesinger
S. S. Schneierson
J. H. Schwab
C. V. Seastone, Jr.
B. C. Seegal
D. Seegal
M. G. Sevag
M. F. Shaffer
H. J. Shaughnessy

M. Shaw
I. L. Shechmeister
A. I. Shelokov
C. C. Shepard
N. P. Sherwood
E. W. Shrigley
G. Shwartzman
G. M. Sickles
G. R. Sickles
M. M. Sigel
M. S. Silverman
J. E. Smadel
D. T. Smith
E. V. Smith
M. H. D. Smith
R. T. Smith
K. C. Smithburn
J. Smolens
J. C. Snyder
M. Solotorovsky
E. H. Spaulding
W. W. Spink
J. Spizizen
J. F. A. Spreat
G. F. Springer
D. H. Sprunt
W. M. Stanley
A. B. Stavitsky
M. Stefanini
A. J. Steigman
G. J. Stein
B. Steinberg
K. Stern
L. A. Sternberger
C. A. Stetson
K. M. Stevens
A. H. Stock
A. V. Stoesser
H. E. Stokinger
G. H. Stollerman
S. H. Stone
C. J. Stormont
C. A. Stuart
J. Y. Sugg
S. E. Sulkin
S. R. Suskind
E. Suter
J. T. Syverton
M. Tager
L. G. Taliaferro
W. H. Taliaferro
D. H. Talmage
I. Tamm
A. R. Taylor
R. M. Taylor
W. H. Telfer
L. Thomas
R. Thompson
R. L. Thompson
W. D. Tigertt
W. S. Tillett
F. B. Traub
H. P. Treffers
L. Tuft
A. Tyler
A. A. Tytell
J. W. Uhr
L. J. Unger
P. L. Varney
J. H. Vaughan
P. Vogel
R. R. Wagner
B. H. Wakeman
D. L. Walker
J. H. Wallace
A. W. Walter
S. T. Walton
M. Walzer
R. Ward
T. G. Ward
W. E. Ward
J. Warren
B. K. Watson
D. W. Watson
R. J. P. Wedgwood
M. Weichsel
W. O. Weigle
A. J. Weil
M. L. Weil
R. S. Weiser
C. Weiss
H. Welch
J. T. Weld
T. H. Weller
E. A. Werber
K. Werman
A. H. Wheeler
M. W. Wheeler
A. S. Wiener
A. T. Wilson
C. L. Wisseman, Jr.
R. W. Wissler
E. Witebsky
F. W. Wittich
H. R. Wolfe
H. F. Wood
W. G. Workman
C.-S. Wright
C. G. Wright
R. H. Yager
Y. Yagi
G. P. Youmans
J. S. Youngner
J. Zichis

RETIRED MEMBERS

S. Bayne-Jones
S. E. Branham
L. Buchbinder
P. F. Clark
G. H. A. Clowes
R. Cole
A. R. Dochez
L. W. Famulener
H. E. Foster
S. B. Hooker
J. A. Kolmer
N. Kopeloff
W. Lintz
A. P. Locke
F. Malzaner
R. R. Mellon
P. K. Olitsky
E. L. Opie
M. C. Pease
E. C. Rosenow
A. H. Sanford
E. W. Schultz
G. W. Wheeler
A. C. Woods

XUM

FE

Sep

FEDERATION PROCEEDINGS *Supplement no. 2*

September 1958

Nutritional Disease

Proceedings of a Conference on Beriberi, Endemic Goiter and Hypovitaminosis A, held at Princeton, N. J., June 1–5, 1958, sponsored by:

THE PATHOLOGY STUDY SECTION, DIVISION OF RESEARCH GRANTS, PUBLIC HEALTH SERVICE

THE WORLD HEALTH ORGANIZATION

THE PAN AMERICAN SANITARY BUREAU

THE FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

and supported by Grant H-3400R, National Heart Institute, National Institutes of Health, Public Health Service

Edited by

THOMAS D. KINNEY AND RICHARD H. FOLLIS, JR.

Published by the

FEDERATION of AMERICAN SOCIETIES for EXPERIMENTAL BIOLOGY

XUM

Federation Proceedings

Published by

FEDERATION *of* AMERICAN SOCIETIES
for EXPERIMENTAL BIOLOGY

American Physiological Society
American Society of Biological Chemists
American Society for Pharmacology and Experimental Therapeutics
American Society for Experimental Pathology
American Institute of Nutrition
American Association of Immunologists

EDITORIAL BOARD

GEORGE M. BRIGGS
HAROLD C. HODGE
CALDERON HOWE
J. F. A. MCMANUS
R. F. PITTS
F. W. PUTNAM
MILTON O. LEE
Managing Editor

FEDERATION PROCEEDINGS is published quarterly. The *March* issue consists of the Abstracts (Part I) of the papers presented at the scientific sessions, and the Program (Part II) of the Annual Meeting of the Federation. The *July* and *December* issues contain symposia and other special papers presented at Federation meetings as selected by the Editorial Board. The *September* issue contains the membership list and other matters pertinent to the Constituent Societies of the Federation.

Entered as second class matter March 16, 1942, at the Post Office at Washington, D. C. under the Act of March 3, 1879. Copyright 1958, by the Federation of American Societies for Experimental Biology. Printed at Waverly Press, Baltimore, Md. All rights reserved.

SUBSCRIPTION PRICE: $8.00 per year ($8.50 Canada, $9.00 foreign) payable in advance. **Single Issues:** *March*, Part I (Abstracts) $5.00, Part II (Program) $1.00; *July* and *December*, $2.00 each; *September*, $3.00. Subscriptions and orders should be sent to:

Federation Proceedings

9650 Wisconsin Avenue **Washington 14, D. C.**

CONTENTS

LIST OF PARTICIPANTS v

PROGRAM vii

INTRODUCTION 1

BERIBERI 3

ENDEMIC GOITER 57

HYPOVITAMINOSIS A 103

RESEARCH AND TRAINING 144

Reprints of sections of this Supplement are not available. Copies of the book may be purchased for $1.50 from FEDERATION PROCEEDINGS, 9650 Wisconsin Ave., Washington, D.C.

Participants

Dr. Stephen B. Andrus—Assistant Professor of Pathology, Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts

Dr. W. R. Aykroyd—Director, Nutrition Division, Food and Agriculture Organization of the United Nations, Viale delle Terme di Caracalla, Rome, Italy

Dr. R. C. Burgess—Chief, Nutrition Section, World Health Organization, Palais des Nations, Geneva, Switzerland

Dr. William J. Darby—Professor of Biochemistry and Director, Division of Nutrition, School of Medicine, Vanderbilt University, Nashville, Tennessee

Dr. Derek Denny-Brown—Putnam Professor of Neurology, Harvard University, Harvard Neurological Unit, Boston City Hospital, Boston, Massachusetts

Dr. Patrick J. Fitzgerald—Professor and Chairman, Department of Pathology, State University of New York, College of Medicine, Brooklyn, New York

Dr. R. H. Follis, Jr.—American Registry of Nutritional Pathology, Armed Forces Institute of Pathology, Washington, D. C.

Dr. Philip Handler—Professor of Biochemistry, Duke University, School of Medicine, Durham, North Carolina

Dr. John Beach Hazard—Chairman, Division of Pathology, Cleveland Clinic, Cleveland, Ohio

Dr. John Higginson—Associate Professor of Pathology and Oncology, University of Kansas, University of Kansas Medical Center, Kansas City, Kansas

Dr. James M. Hundley—FAO Nutrition Advisor to UNICEF, Food and Agriculture Organization, United Nations, New York, New York

Dr. Elsa Orent Keiles—Executive Secretary, Metabolism and Nutrition Study Section, Division of Research Grants, National Institutes of Health, Bethesda, Maryland

Dr. Thomas D. Kinney—Professor of Pathology, Western Reserve University School of Medicine, Cleveland, Ohio

Dr. Cosmo G. Mackenzie—Professor of Biochemistry, University of Colorado Medical School, Denver, Colorado

Dr. Karl E. Mason—Professor and Chairman, Department of Anatomy, University of Rochester School of Medicine and Dentistry, Rochester, New York

Dr. Josip Matovinovic—Docent, Division of Endocrinology and Metabolism, Interna Klinika, Medicinski Fakultet, Zagreb, Yugoslavia

Dr. D. S. McLaren—Medical Research Officer, East African Institute for Medical Research, P.O. Box 162, Mwanza, Tanganyika

Dr. Henry D. Moon—Chairman, Department of Pathology, University of California School of Medicine, San Francisco, California

Dr. Eli M. Nadel—Executive Secretary, Pathology Study Section, Division of Research Grants, National Institutes of Health, Bethesda, Maryland

Dr. Robert E. Olson—Professor of Biochemistry and Nutrition, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania

Dr. H. A. P. C. Oomen—Nutrition Section, Institute of Tropical Hygiene, Mauritskade 57, Amsterdam, Holland

Dr. V. N. Patwardhan—Director, Nutrition Research Laboratories, Indian Council of Medical Research, Coonoor, South India, India

Dr. B. S. Platt—Director, Human Nutrition Research Unit and Head, Department of Human Nutrition, London School of Hygiene and Tropical Medicine, Medical Research Council and University of London, Mill Hill, London N W 7, England

Dr. V. Ramalingaswami—Research Professor of Pathology, All India Institute of Medical Sciences, New Delhi, India

Dr. Arnold E. Schaefer—Executive Director, Interdepartmental Committee on Nutrition for National Defense, National Institutes of Health, Bethesda, Maryland

Dr. Nevin S. Scrimshaw—Regional Advisor on Nutrition, Pan American Sanitary Bureau, Regional Office for the Americas, WHO, and Director, Institute of Nutri-

tion of Central America and Panama (INCAP), Carretera Roosevelt, Zona 11, Guatemala, Central America

Dr. John B. Stanbury—Assistant Professor of Medicine, Harvard Medical School, Associate Physician, Massachusetts General Hospital, Boston, Massachusetts

Dr. Frederick J. Stare—Professor of Nutrition and Head, Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts

Dr. Carlos Tejada—Chief, Pathology Section, Instituto de Nutricion de Centro America and Panama (INCAP), Guatemala City, Central America

Dr. Erwin A. Uehlinger—Professor of Pathology, Medical Faculty, University of Zurich, Institute of Pathology, Schmelzbergstrasse 12, Zurich, Switzerland

Program

June 2, 1958

BERIBERI

Chairman: Dr. Burgess

I. Epidemiology ..... *Dr. Burgess*
II. Clinical Features of Endemic Beriberi ..... *Dr. Platt*
III. The Pathology of Endemic Beriberi ..... *Dr. Follis*
IV. Experimental Thiamine Deficiency ..... *Dr. Follis*
V. The Metabolism of Thiamine ..... *Dr. Handler*
VI. Special Problems Concerning Beriberi
  A. neurological ..... *Dr. Denny-Brown*
  B. infantile beriberi ..... *Dr. Burgess*
  C. rice ..... *Dr. Aykroyd*
VII. The Pathogenesis of Beriberi ..... *Dr. Follis*

June 3, 1958

ENDEMIC GOITER

Chairman: Dr. Scrimshaw

I. Epidemiology ..... *Dr. Scrimshaw*
II. Clinical Aspects ..... *Dr. Matovinovic*
III. Pathology
  A. the pathologic anatomy of endemic goiter before the introduction of iodinized salt .. *Dr. Uehlinger*
  B. the influence of iodine on Swiss goiter ..... *Dr. Uehlinger*
  C. the relation of thyroid changes to nutritional status ..... *Dr. Tejada*
IV. Experimental Goiter ..... *Dr. Mackenzie*
V. Iodine Metabolism ..... *Dr. Stanbury*
VI. The Pathogenesis of Endemic Goiter ..... *Dr. Hazard*

June 4, 1958

HYPOVITAMINOSIS A

Chairman: Dr. Aykroyd

I. Epidemiology ..... *Dr. Patwardhan*
II. Clinical Aspects ..... *Dr. Oomen*
III. Pathology
A. experimental ..... *Dr. Mason*
B. man ..... *Dr. Ramalingaswami*
IV. Biochemical Considerations ..... *Dr. Handler*
V. Pathogenesis ..... *Dr. McLaren*
VI. Summary ..... *Dr. Aykroyd*

June 5, 1958

RESEARCH AND TRAINING IN NUTRITION

Chairman: Dr. Kinney

I. Suggested Needs for Research on:
A. beriberi
B. endemic goiter
C. hypovitaminosis A
II. Training and Research
A. general remarks
B. clinical and laboratory
C. field
III. Recommendations of Conference

INTRODUCTION

AT A REGULAR MEETING in September 1956, the Pathology Study Section, Division of Research Grants of the National Institutes of Health, United States Public Health Service, under the chairmanship of Dr. R. E. Stowell, discussed certain areas in pathology which might be in need of more vigorous development as far as research and training facilities were concerned. One such underdeveloped area appeared to be nutritional disease. On the basis of this Drs. Kinney and Follis were asked to explore the matter with the possible view of holding a conference dealing with problems associated with nutritional disease. After preliminary consultations, it was recommended that the Pathology Study Section approve a proposal to hold a conference dealing with certain specific disease syndromes which are prevalent throughout the world today. These diseases could then be used as prototypes with which to explore further directions for research and training facilities in this country and abroad. This proposal was recommended for approval by the National Advisory Heart Council and the National Advisory Arthritis and Metabolic Disease Council. A grant was awarded by the National Heart Institute to defray the costs of holding a conference and publishing its transactions.

It was the consensus of the planning group that there are today a number of specific deficiency disease syndromes which have world-wide prevalence. Although such diseases can be treated or prevented precise knowledge of their prevalence or pathogenesis is lacking. From the publications of the World Health Organization-Food and Agriculture Administration Joint Conferences on Nutrition it was apparent that, excluding kwashiorkor, on which a number of conferences have recently been held, there were three important entities which could profitably be discussed. These were beriberi, endemic goiter and xerophthalmia (hypovitaminosis A). Since these three syndromes are seldom seen in this country, it was obviously necessary to invite individuals from abroad who had first-hand experience with these diseases. We were fortunate in gaining the cooperation of the World Health Organization, the Pan American Sanitary Bureau and the Food and Agriculture Organization of the United Nations. All three agreed to act with the Pathology Study Section as co-sponsors of the conference. The participants were then invited on the basis of their knowledge of the entities to be discussed and because of their interest in research and training in nutritional disease. It was agreed that the conference should be a small one of the round-table type with a minimum of formal presentations and a maximum of discussion. These Transactions represent the words of the participants which were recorded and transcribed, and then edited by each individual while the conference was in session. The Editors assume responsibility for those deletions which were necessary for the sake of clarity and continuity.

On behalf of the Pathology Study Section the undersigned wish to thank all who participated in the conference and particularly those who gave so much help during the planning sessions, namely Drs. Philip Handler, Cosmo G. Mackenzie, Mark Hegsted, J. M. Hundley and Elsa Keiles.

THOMAS D. KINNEY
RICHARD H. FOLLIS, JR.

BERIBERI

I. Epidemiology

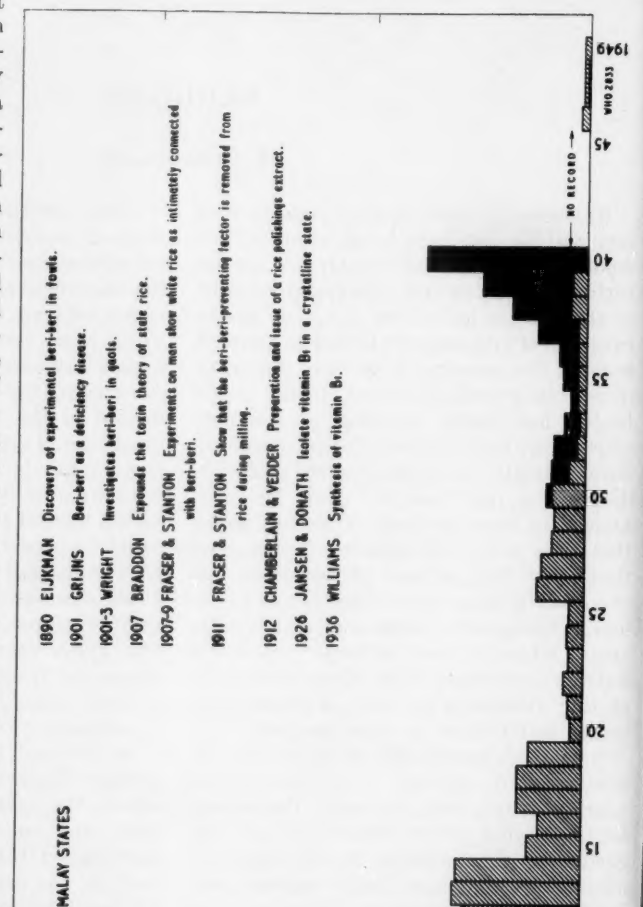
Burgess: To most medical men, beriberi is something they have heard about in their student days, important mainly because the early observations and subsequent research in this disease led to the discovery of the existence of vitamins and helped to establish some of the principal features of deficiency disease in general. However, to the public health nutritionist working in countries where raw, highly-milled rice is consumed, beriberi is still one of the unsolved problems, despite the fact that the cause has been known for some 40 years. Time has shown that it is not a disappearing disease, and that only the pattern of incidence has changed. In some areas, where it was prevalent, it has largely disappeared, and in other areas, where it was hitherto practically unknown, more and more cases are now occurring. Beriberi is therefore of considerable health significance in some countries and, although extensively studied in the first two decades of this century, it has not received nearly as much attention since. The reasons for this neglect are not difficult to find. The decline in the incidence in the originally affected urban areas probably created a feeling that the disease was no longer of major public health significance. Also, in areas where the deficiency is less acute and medical services limited, beriberi can cause many deaths without the existence of widespread disease being recognized. These two points will be discussed later. In addition, in most countries in which beriberi occurs, communicable disease is also present. The training of the majority of health officers has been in the recognition and control of these latter conditions, and attention has been therefore largely concentrated on them. This is still true in many parts of the world, and if there is to be reasonable progress in the prevention of nutritional diseases generally, some reorientation in the training of medical and health personnel is necessary.

There are two main approaches to the study of any disease: the study of the individual case, and the epidemiological study of the occurrence of the disease and the factors which influence it. It is the latter approach which is my concern. It seemed to me that a good way to open this discussion is to give some account of the changing pattern of this disease in the last 5 or 6 decades. This graph (fig. 1) shows figures for the admission rate to hospitals per thousand population in Malaya, a country where I myself worked for 22 years; hence, I understand the economic and other changes which have influenced the pattern of the disease. I will discuss these in some detail because they bring out some important points. In this graph there are also figures for the disease in Hong Kong which show a very different trend, which I think is of some importance.

In Malaya there are three rice-eating groups. There are the indigenous Malays, who in the early days lived mostly near the coast and cultivated their own rice and hand-milled it. In the latter part of the 19th century and early decades of this one, the Chinese immigrated in great numbers. They were tin miners whose food was imported into the country and who preferred a highly milled rice. Lastly, when the rubber industry began to open up, large numbers of southern Indians were brought in to work on rubber estates. Their food was also imported and they preferred parboiled rice. The differences in the incidence of the disease in these communities in the early days were investigated, and the findings helped considerably in establishing their exact nature. As a result we have some excellent accounts of the disease 50 or 60 years ago.

At that time, beriberi was responsible for enormous morbidity and mortality among young able-bodied Chinese laborers. I doubt if the disease was ever seen in a more severe

form than in the latter part of the last century and in the beginning of this century in Malaya, and it may be worthwhile to give a brief account of the conditions under which it occurred. The Chinese worked in mines which were often in remote jungle areas with poor means of communication and little or no local production of food. As all the food had to be transported, the range of choice was narrow. The system of recruitment and employment of labor to work these mines had a profound influence on the incidence of beriberi. Laborers were brought in under contract. They were recruited in China, given a sum of money, transported to Malaya, lodged in large huts, and provided with bedding, a mosquito net and food for the tenure of their agreement, which might extend from 6 months to 2 years. They were known as 'Sinkhehs', a term generally used in Malaya, meaning a newcomer. After they had completed their agreement, they were described as 'Laukhehs', or old-timers, and were paid wages; beyond being provided with a roof over their heads, they organized their lives in their own way. There was also a third group in these mines: owners, foremen, etc., who were better off financially. Braddon (1) gives a very interesting account of the meals eaten by 'Sinkhehs in 1907, who got unlimited quantities of rice as part of their wages. He describes the meals of the day as follows: "A large


ADMISSIONS TO HOSPITALS - FEDERATED MALAY STATES
ADMISSIONS TO HOSPITALS - HONG KONG
1890 EIJKMAN Discovery of experimental beri-beri in fowls.
1901 GRIJNS Beri-beri as a deficiency disease.
1901-3 WRIGHT Investigates beri-beri in gaols.
1907 BRADDON Expounds the toxin theory of state rice.
1907-9 FRASER & STANTON Experiments on man show white rice as intimately connected with beri-beri.
1911 FRASER & STANTON Show that the beri-beri-preventing factor is removed from rice during milling.
1912 CHAMBERLAIN & VEDDER Preparation and issue of a rice polishings extract.
1926 JANSEN & DONATH Isolate vitamin B1 in a crystalline state.
1936 WILLIAMS Synthesis of vitamin B1.
NO RECORD
PER MILLE
0
10
20
1895
1900
05
10
15
20
25
30
35
40
45
1949

bowl of rice in the form of gruel was consumed in turning out to work at 5:30 in the morning. The next meal, again rice with a few vegetables, was eaten upon return from work at 11 A.M., and another meal of the same nature was eaten at 1:00 P.M. The principal meal of the day, again rice with small quantities of other food, was taken at 5 or 6 P.M. In addition to this, the laborer often got up during the night to eat extra meals. On the average, mining laborers ate a quantity of rice weighing between one and one-half and two pounds each day." They were, therefore, getting between 2,500 and 3,200 calories from rice alone, and were therefore very likely to suffer from beriberi. Hamilton Wright (2) records that 800 out of 2,400 such recruits died of beriberi at one isolated mine in a 2-year period. The 'Laukheh' or old-timer, on the other hand, purchased his own food, and his diet changed somewhat as a result. If he remained in an isolated mine, the range of foods he could obtain was narrow, and he still was prone to the disease. However, if he moved to the rapidly growing towns and worked in shops, pulled rickshaws or did other work of that sort, he contracted the disease much less frequently. The relatively well-to-do mine owner seldom got the disease.

Among the Chinese population as a whole the disease was very common, and in Kuala Lumpur, now the capital of the Federation of Malaya, originally a mining town, there was at the turn of the century a hospital with 14 wards, of which 8 were reserved for beriberi cases. Braddon (1) estimated that of every thousand Chinese in the country at that time 120 got beriberi annually, 80 had it severely, and 16 fatally. The very marked reduction of the admission rate to the hospital from 1900 to 1920 is striking. The reasons for it are changes in living conditions. In the early 1900's, there were four to five men to every woman and a very high proportion of the men were in labor camps. As time went on, women came, increasing in numbers so that the sex ratio leveled off. Men married; homes were set up, and as women demanded a greater variety of food, local vegetable gardens were developed. Also, roads were built which reached most of the remote mines, so that the transfer of food became easier, and the degree of imbalance in respect to thiamine was greatly decreased. Cases of the disease still occurred at the rate of one in two admissions to the hospital per thousand population. You will recognize this does not represent the true number of beriberi cases. The majority of sufferers were likely to have only a chronic, mild neuropathy, and never reached hospitals, so that these figures only give a relative idea of the state of affairs. And as we will discuss later on today, it seems likely that even under these conditions there can be quite a high incidence of infantile beriberi. The graph (fig. 1) shows two periods during which there was an increased incidence, between 1925 and 1930 and once again in 1938 and 1939. The economy of Malaya depends almost entirely on rubber and tin, and the price of both of these commodities was booming during these two periods. In all the medical reports at that time, there is constant reference to the fact that with improved economic conditions there is more beriberi. It is also worth noting at the same time that there was also an increased infantile mortality associated with increased prosperity.

During these periods the disease became prevalent among small holders who owned an acre or two of rubber land, as well as small areas of rice fields. When rubber sold for a good price, they sold their paddy stand and purchased the greatly preferred highly milled Siam rice. When the return for rubber was low, the small holder had to economize, and he therefore hand-pounded and consumed his own rice. Prosperity, therefore, introduced the disease into communities where under poorer circumstances it did not exist, or was at least relatively rare. A similar increase in beriberi is occurring today in rice-growing communities in certain other countries as a result of the introduction of small power mills.

In the Hong Kong figures (fig. 1) there is a reverse trend. In 1936 conditions were comparable with those in Malaya, a rice-eating community fairly well stabilized. And it may be, too, that during the war hunger

FIG. 1. The prevalence of beriberi in hospital admissions, 1895–1949, Federated Malay States and Hong Kong.

made people consume food which they had not previously found acceptable, and this brought about a permanent change in dietary practice. Lastly, thiamine is now cheap and readily available and early cases are treated; this results in fewer admissions to hospitals. Beriberi doesn't appear to be a major public health problem in either of these countries today.

After the war there was a very marked decrease in the incidence of beriberi. The reasons for this are not properly understood, but there are certain changes that probably account for it. There was a marked shortage of rice after the war and wheat was brought in to replace rice. The cost of rice relative to other foodstuffs was higher than it had been pre-war. It also appears likely that the total consumption of rice decreased.

In other countries in Southeast Asia, however, the disease is becoming much more frequent. With increasing prosperity and sophistication the practice of hand-milling is giving way to machine milling, and rice mills are rapidly spreading into rural areas. When the small farmer himself consumes a

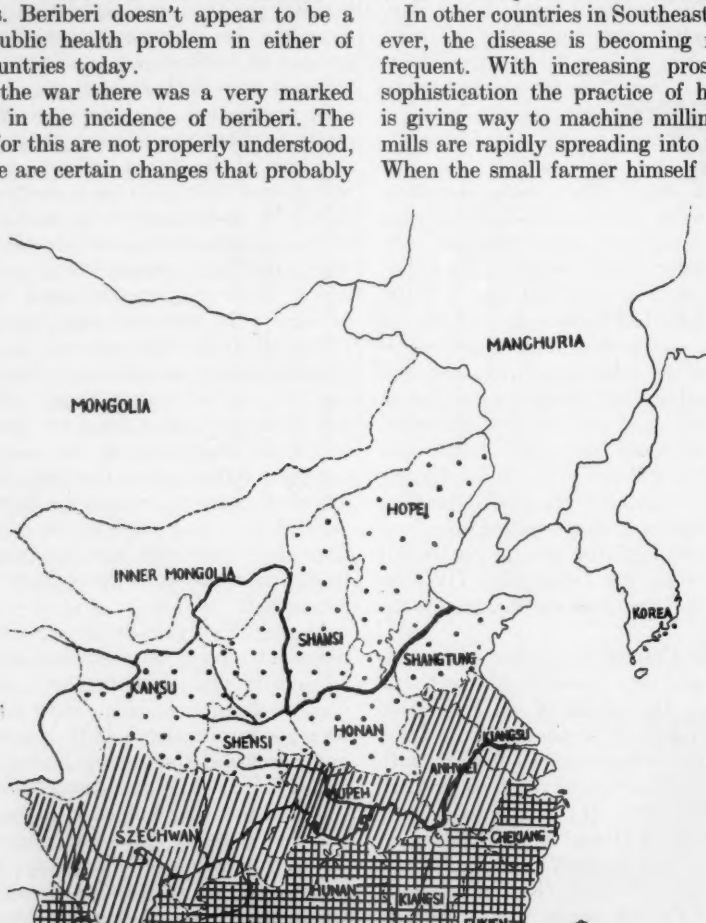


FIG. 2. Beriberi in China, 1933–34

highly-milled rice, it seems likely, in the light of past experience, that there will be an increased incidence of beriberi. He is usually also, of course, a very big consumer of rice, since he has plentiful supplies, and on the whole the range of other foodstuffs he consumes is narrow and the amounts are small. Only limited surveys have so far been carried out in these countries; these indicate that in some areas at least there is a high incidence of the disease. Ramalingaswami carried out one of these surveys in North Thailand, and in one area he found some 24% of the adult population had neuropathies attributable to beriberi. We have other reports, though in no detail, of the occurrence of cases 3 to 4 months after a new mill is introduced. I have had, recently, an account of a mill being constructed in a village, and after a period of a few months the head man closed it down because of the amount of ill-health which he attributed to it. There appears to be little doubt that beriberi is still an important disease, but it is clear that a great deal more information is needed.

PLATT: I want to report some data collected by Dr. H. S. Gear on the prevalence of beriberi in China in 1933–4. Three areas of different density are shown on the accompanying map (fig. 2), the area most affected being in the south; the Yangtze Valley is the area in which the disease is fairly common, and in the northern provinces cases are least common. These data should be examined in relation to the type of agricultural areas which are given in Table 1, together with estimates of caloric intake and vitamin B_1 supply. The association between the vitamin B_1 supply and the frequency of occurrence of beriberi is manifest.

Gear also determined a sex incidence in the hospital data, using a basis of 2 males to 1 female as an index of the general hospital population ratio. There were 1,252 males to 24 females, and combining the data for the two years 1933–4 showed a remarkable incidence of a male susceptibility and concentration, particularly in young adult males.

It might be noted that the Chinese have recognized beriberi as a clinical entity for many centuries.

RAMALINGASWAMI: I should like to make a comment on the relationship between the incidence of beriberi and rice mills. As Burgess mentioned, there are innumerable references indicating the rise in the incidence of beriberi following introduction of small Engleberg-type rice mills into a community. In the course of our survey in Thailand, this was very clearly brought out to us when we looked at the situation in two areas, one in the north and the other in the northeast of

TABLE 1. VITAMIN B_1 IN THE FOOD IN THE AREAS LISTED IN TERMS OF INTERNATIONAL UNITS OF VITAMIN PER ADULT MALE UNIT PER DAY

Agricultural Area	Provinces Included	Est. Calorie Intake	No. Units from Sources other than Rice	No. Units from Rice	Est. Vitamin B_1 Intake, IU
Double Cropping rice	Kuantung Kuangsi Fukien	3403	77	173	250
Rice-tea	Kiangsi Hunan Chekiang (part) Anhwei (part)	2507	159	107	266
Yangtse rice wheat	Kiangsi Chekiang (part) Anhwei (part) Hupeh	3843	95	187	282
Szechuan rice	Szechuan	3520	132	190	322
South western rice	Kweichow Yunnan	4121	172	197	370
Spring wheat	Kansu Shansi (part) Shensi (part) Suiyan Tsinghai Ningsia	3783			455
Winter wheat Kaoliang	Shantung Hopeh (part) Anhwei (part)	4484			675
Winter wheat millet	Hopeh (part) Honan Kansi Shansi (part) Shensi (part)	3842			687

that country. In the former area there was one rice mill for every 8,700 persons, whereas in the latter area there was one for every 13,000 persons. The difference in the incidence of peripheral neuropathy in the two areas, especially in the expectant and lactating women, was quite striking. In the area with more rice mills, the incidence of neuropathy was almost twice that of the other.

Another finding to which I would like to refer concerns the seasonal incidence of beriberi. The disease increases during the time of harvest. The reasons are that during harvest there is increased physical exertion, and more rice is eaten in order to meet the caloric requirements.

FOLLIS: What is the current status of the availability of data on the prevalence of beriberi, both in hospital populations and the population at large in areas where the disease may be expected to occur today? That is, what sort of data do we have for 1956 or 1957?

BURGESS: Very little information, indeed. We've had some work done among lactating women, and we find, for example, in a selected group from a child health center anything from 25 to 50% of the women are recorded as showing some chronic neuropathies. But no general survey has been done.

I'd like to ask Oomen to say something about the change in the incidence of beriberi in Java and in Asia generally since the war. We were told when we were in Asia that beriberi has practically disappeared, perhaps as a consequence of a shift from imported rice to a greater use of home-pounded rice in Java. Since you have been there for a considerable period of time and know the country intimately, will you comment on this and indicate your experience and impressions?

OOMEN: As a matter of fact, I encountered very few cases of beriberi in Indonesia after the war in a big hospital, but I saw many cases in a small hospital before the war. I think this is a fairly general experience in Indonesia, but it is a large country with contrasting living conditions. On the whole, I think that beriberi as a health problem is nearly finished. I have never experienced beriberi in Indonesia on a scale as it was described between the years 1900 and 1920. I think you could say that in Indonesia beriberi vanished at the mere mention of the vitamin. But do not forget that it was a condition especially affecting closed or controlled communities.

As to the question of the difference between home-pounded rice and milled rice, I think nowadays most of the rice consumed in Java is milled rice, but the degree of milling is not too high. I am not quite certain about the administrative mechanism, but there is a regulation requiring that the rice on the market be not very highly milled. In small villages the rice is still home-pounded and it is then understandable that you do not meet cases of beriberi in rural surroundings.

During Burgess' talk, I was reminded of the conditions existing in West Borneo as being very comparable with those in Malaya. There you have a Chinese population which is very fond of white rice.

II. Clinical Features of Endemic Beriberi

PLATT: The task assigned to me is "to develop the highlights of what endemic beriberi was clinically". For me, this means looking back 20–25 years to a period from February 1933 to September 1937 when I was working in Shanghai almost exclusively on beriberi. My research came abruptly to an end with the extension of the Sino-Japanese War to the Shanghai area. This incident resulted in the loss of many records as well as the cessation of several studies before they were complete.

This work on beriberi was my introduction to research in nutrition. I selected the subject for two reasons: *a*) the disease was of common occurrence, and *b*) patients with beriberi had a pronounced creatinuria—a phenomenon in which I was interested at the time. In the text books the statement was commonly made that creatine was not found in the urine of normal adult males, and only intermittently in urine of females. It was found (3) that creatinuria occurred in apparently healthy normal Chinese male adults. A marked creatinuria was frequently encountered amongst male Chinese out-

patients; almost all these patients were found to be suffering from beriberi and, on recovery from the disease, the degree of creatinuria was progressively reduced; this sign was, in fact, used for some time as an index of recovery. The increased excretion of creatine in the urine of the more severe cases of beriberi has been mentioned (4), as has the fact that it was this feature that first directed our attention to the disease (5). I suggest it is an observation that would merit further investigation.

I would like briefly to consider the forms of beriberi in relation to age and sex. Where it occurs in child-bearing women, the disease presents features of special interest which were dramatic in the years 1939, 1940 and 1941 in Hong Kong and are fully described by King and Ride (6). I cannot do justice to this investigation in a brief summary and I shall select only one point for emphasis —the one considered by King and Ride to be the most interesting feature of the series of 37 cases of beriberi complicating pregnancy; i.e. "the fact that 70% of the patients with beriberi showed co-existing evidence of pregnancy toxemia".

Infantile beriberi will be taken up later today. I want to draw attention to the similarity between the accumulation of carbonyl compounds in a case of infantile beriberi (case no. 2472, table 4 *A* (5)) and in cases of acute beriberi of adults. This is also relevant in view of the possible role of carbonyl compounds, more particularly methyl glyoxal, in the development of the syndrome of infantile beriberi, of the evidence for the occurrence of this substance in human breast milk showing no peroxidase activity and of abnormal amounts of 'pyruvic acid' in the milk of a vitamin B_1-deficient wet nurse (5).

One of my most vivid recollections of beriberi in the young was of a grossly edematous toddler who responded to treatment with vitamin B_1 and proved to be extremely marasmic; this was also my first experience (in 1935) of protein malnutrition complicated by thiamine deficiency.

Beriberi in male adults, non-pregnant or lactating females, and older children and adolescents may be of three main types; they are *a*) the chronic, dry, atrophic type, *b*) the mild and subacute forms and *c*) the acute fulminating type. The types *a*) and *c*) together were only about 10% of all the cases seen of these three types in hospital practice in Shanghai (7).

The chronic, dry, atrophic type—with wrist-drop and foot-drop—is generally found only in the older adult. None show the biochemical changes characteristic of the acute type of beriberi. Beriberi is to be found in the chronic phase of the disease and there is little or no response to long, continued treatment by dietary or other measures, including physiotherapy. There is evidence for the association of this form of beriberi with consumption of alcohol, with lead poisoning from drinking Chinese rice wine warmed in pewter pots or with lead from other sources, and it has also been associated with opium addiction (8). Chronicity appears to be the key to the development of this type of beriberi. Lim (9) remarks that signs of the dry atrophic type of beriberi would rapidly begin to appear if the diet of a patient in the convalescent period following the acute phase of beriberi were cut down to rice, salted vegetables and salted eggs. Rombergism is occasionally seen in association with other signs of chronic involvement of the nervous system.

I shall now discuss some features of the mild and subacute cases of beriberi. The nervous manifestations include alterations in tendon reflexes. Paresthesias are common. The limbs or the parts of the limbs affected appear to be determined by a combination of three factors; the length of the nerve, the amount of work done by the limb or limbs, and possibly by the blood supply. Men of the laboring class usually complain first of disturbances in the legs below the knees; women complain much more frequently of sensations in their fingers. When the lesions are well developed, occupational factors are revealed; e.g. 'thumb-drop' may develop in the right hand of a tailor's cutter, and a man stripping hides will show more pronounced changes in the left arm with which he wields his knife than in the right arm used in holding the hide. When a vocal cord is affected, it will be found to be the left one, i.e. the one supplied by the longer of the two laryngeal nerves.

Complaints about the calf muscles are interesting; they are generally of a feeling

of fulness or tightening of the muscles—and it is this sensation and the pain which develops on walking rather than muscle weakness which tends to immobilize the patient. These 'fatigue pains' resemble the pain of muscle ischemia. Muscle cramps occur—especially at night before the patient goes off to sleep. Wenckebach's description of skeletal muscle is interesting: "I persuaded one of my colleagues in Java to remove a small piece of muscle from one of these distended calves. There was neither a flow of blood nor edema present. The wound remained dry. The muscle-bundles protruded out of the opening in the thin fascia. After this the patient was put under the usual treatment. The next day the calf was somewhat softer and soon returned to normal. A week later the other calf of this patient was opened and blood and edema escaped freely" (10). This 'hardening' of the calf muscles is of value in detecting beriberi in its early stages; in a later stage there may be pitting edema and fluid may be obtained by acupuncture. The edema that occurs in these mild and subacute cases is well known by the patient to be related to physical activity and to appear at the end of a day's work.

The signs and symptoms related to the cardiovascular system are prominent and important; they range from complaints of breathlessness on exertion and palpitation to those found in the acute fulminating form (the Shöshin of the Japanese). In some of the subacute cases it is likely that the edema is, in part, due to some degree of congestive heart failure. In well-developed cases, tachycardia is always present. The heart is enlarged in established cases. Aalsmeer and Wenckebach (11) are of the opinion that cardiac dilatation may be detected long before there are any signs of polyneuritis; this, in my experience, is not always true. The physical signs of involvement of the heart and circulation have been fully described by Wenckebach (10).

The onset of the presenting symptoms in about half the patients with the subacute type of beriberi is associated with a nonspecific bout of fever. This is important since, as will be shown later, the pyrexial state is accompanied by an increase in the need for thiamine.

Another feature of this group of subacute beriberi patients is that they usually show evidences of insufficiency of other food factors, including ascorbic acid (12), riboflavin, niacin and vitamin A. There is little doubt from the knowledge we now have that the youths we saw also suffered from protein malnutrition. Almost all of them had chronic multiple ulcers of the legs of the type suspected to be associated with protein insufficiency (13). It is of interest to note in this connection that young factory apprentices in Peking had been shown to have a low urea N:total N in their urines; a low value for this ratio is indicative of protein malnutrition (14).

Many of the patients studied in this group of mild and subacute beriberi were apprentices in small factories in Shanghai. Some observations made in a feeding experiment in five such factories where flash lamp bulbs were made are summarized in table 2.

The employees in Foo Lih were fed from a kitchen run under my supervision for a year. The main difference in the food they had and that in the other factories was that the rice we provided was milled from paddy each day with an Engleburg huller; the milled rice was cooked without washing and in such a manner that no thiamine was lost in the water added during cooking. Treated in this way, rice retains, when cooked, about 80% of the thiamine in the original grain. The rice fed to the employees in the other factories was provided by food contractors; it was often grain that had been stored as highly-milled grain, sometimes for long periods, when it became infected with a mold which matted the grains together into large lumps. These had to be broken down, remilled and washed thoroughly to render it edible. Most of the thiamine in the original grain is lost by such a treatment. The results in the table show that about three-quarters of the employees in the contractor-fed factories either had edema or had been sent back to their villages, sick. Among those in the factory who had been fed on rice prepared in effect as nearly as possible in the same manner as in their rural homes there were only two cases with edema; none had been sent home.

The behavior of the youths fed on the 'country' rice was remarkably different

TABLE 2. TABLE SHOWING EFFECT OF FEEDING RICE OF HIGH VITAMIN B_1 CONTENT ON INCIDENCE OF EDEMA AND ON LABOR TURNOVER IN SHANGHAI FACTORY APPRENTICES

Factory	Date of Visit	No. Subjects	No. in Age Groups			No. with Edema of Legs	No. Having Left since Last visit	No. with Edema or Left Factory / No. at Previous Visit × 100
			10-yr.	15-yr.	20+ yr.			
Foo* Lih	7.10.36	38	18	20		15		
	18.6.37	53	22	27	4	1		
	15.7.37	53				2		2
Kwen Ming	21.1.37	42	16	26		13		
	23.6.37	63	26	37		11		
	6.7.37	48				16	15	65
Yung Lih	18.6.37	49	33	14	2	20		
	29.7.37	25				10	24	70
Ying Poo	7.7.37	49				24		
	29.7.37	39				29	9	78
San San	11.6.37	61	12	27	1	2		
	15.7.37	41				25	20	74

* Factory fed from experimental kitchen supplying rice freshly milled from paddy and cooked without washing.

from that of the ones from the contractor-fed factories. The latter were, as described above, listless and apathetic. Those in the Foo Lih factory became noisy, mischievous and high-spirited. This was one of our studies that was not completed; the area in which the factories were situated was one of the main battle zones.

The acute fulminating form of beriberi, though relatively uncommon (about 5% of the total cases seen), was most interesting in many respects. One feature common to all patients with this form of beriberi was that until early in 1936, when I obtained from Dr. A. G. Van Veen nearly 0.5 gm of natural crystalline vitamin B_1, all died within a day or two after admission. Following dosage with a few milligrams of the vitamin, 17 of a series of 21 cases recovered, many of them within a few hours. The experience of Hawes, Monteiro and Smith (15) confirmed mine; they reported only 23 deaths in a series of 100 severe cases. This was in contrast with their earlier experience when they lost 80 to 90% of the same type of case within 1 to 12 hours after admission, whatever variety of treatment was given.

Hawes *et al.* (15) quote de Langen's description (16) as being the best in the English language. Since it may not be readily accessible to readers, it is quoted:

"The whole picture here is dominated by the insufficiency of the heart and vessels, and usually runs a fatal course. The milder attacks may pass over to this type, but as a rule pernicious beriberi breaks out suddenly in a peracute form, before any other manifest symptom has proclaimed the presence of beriberi in the patient. When the nerve lesions develop early, we do not see Shōshin; the heart is saved from this extreme insufficiency by the patient's being forced to a complete rest at an early stage of his attack. If the nerve symptoms come on late, and the patient with his labile heart and vessels can remain for some time on his feet, and must often even continue to work in order to earn his daily bread, the best possible conditions are present for the sudden development of Shoshin. Patients are sometimes known to die of this form of beriberi when next to no oedemata are as yet demonstrable.

"The death from Shōshin beriberi is often very terrible. The patients are severely dyspnoeic. Violent palpitations of the heart allow them no moment of rest. An intense precordial agony is often one of the most distressing symptoms. The patient is very restless; he cannot lie still for an instant, but tosses and turns about violently from side to side in bed the whole time. The precordial oppression is often felt in the epigastrium. It is a feeling of heaviness, constriction and oppression, sometimes of actual pain. The patient moans and shrieks, and his cries take on a rather special character because of the coincident hoarseness or sometimes aphonia. He is often intensely thirsty, but reacts to drinking by vomiting. He has an anxious look on his face. His pupils are dilated; his respirations frequent and superficial.

"In spite of his dyspnoea his accessory muscles of respiration are brought but little into action, often not at all. He does not want to sit up in bed and help his respirations with both hands as is the case with other dyspnoeic patients, but, so far as his restlessness will permit it, he prefers to lie flat in bed. He does not cough or spit. In the heart region, the epigastrium and the neck, widespread and powerfully undulating pulsations are to be seen. There is an obvious cyanosis, more marked during inspiration. The pulse is fuller than might

be expected, is usually regular, even and weak, with a frequency of from 120 to 150 per minute. A very high frequency of the heart is seldom seen. Over the heart area a wave-like motion may be felt. The liver is enlarged and tender. The epigastric region is spontaneously painful. Percussion shows the heart to be enlarged in all directions, but mostly to the right. The broadening of the area of aortic dullness, caused by the vascular stem, is plainly demonstrable extending into the second intercostal space to the left. Changes in position affect the heart outline markedly; it conforms to the position assumed by the patient. Over the body of the organ systolic murmurs may sometimes be heard, but not always. The heart action may only be described as tumultuous, although the sounds are not so powerful as in the last type of the disease described. Murmurs and sounds are difficult to localize. Vessel sounds are heard not only over the femoral artery, but also over the vessels of the elbow. These may become so loud that, as Shimazono has reported, they may even be heard without the aid of a stethoscope and in some instances even at a distance from the bed. The systolic pressure, which at first remains normal, finally falls slowly to below 100, often reaching 80 or even 70.

"Strangely enough there is but little disturbance to be found in the lungs; at most there is a mild tympany on percussion. Just before death there tends to be a slight dullness at the lower borders. The respiration is rough and whistling, but without rales. As the condition becomes worse, rales may appear, at first dry and high-pitched, but soon becoming more moist. At the same time the pulse becomes thinner, the veins dilate more and more and the patient dies intensely dyspnoeic, but usually fully conscious. It is but seldom that a patient survives an attack, but even if we happen to be fortunate enough to bring him into a less immediately threatening condition, the risk of a sudden relapse is never absent." (16).

I intended to imply by my statement that patients exhibiting this form of beriberi had one feature in common in that there were also differences. One of the most conspicuous differences is variability of the edema—in degree and extent. Some patients show very little, and this is a major difficulty in the way of classifying beriberi as is conventionally done—into two forms, wet and dry, the wet form comprising the more or less acute forms. Hawes *et al.* (15) state that "gross oedema on which so much stress has been laid has no relation whatsoever to the severity of the case." I believe that the onset of the acute phase of the florid form of the disease may be so sudden that there is no time for what J. P. Peters (17) calls 'pool formation' to occur. It is by no means clear what the disturbances in the body water relationships are in thiamine deficiency, but they involve at various times, among other factors, imbibition, heart

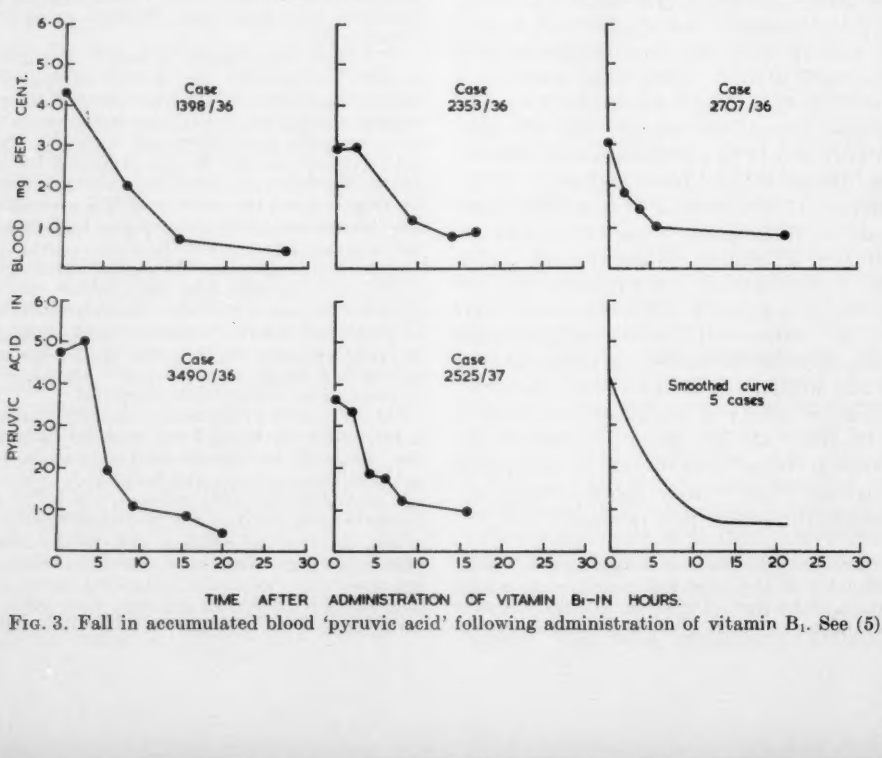


FIG. 3. Fall in accumulated blood 'pyruvic acid' following administration of vitamin B_1. See (5).

failure and impaired renal function. There is in the kidney a biochemical lesion of the type found in the brain (18) and it is not uncommon to find albumin and casts in the urine of beriberi patients and to find that these changes disappear in a few days after treatment with vitamin B_1. There is often a rise in non-protein nitrogenous substances in blood to about three times the normal levels (19).

Vomiting is a common event in the acute case; the effects of this may help to explain the differences between the values obtained for carbonyl compounds in body fluids by the two methods which furnish values for bisulphite-binding substances (B.B.S.) and 'pyruvic acid'.

Undoubtedly, the availability of the vitamin and the results of the researches of R. A. Peters and his associates revolutionized the outlook on beriberi around 1935–6. I have always fully recognized our indebtedness to these facts (20), but it is only fair to state that in the printed Proceedings of the Physiological Section of the Third General Conference of the Chinese Medical Association held at Canton in November 1935, Platt and Lu (19) reported the presence of bisulphite binding substances (B.B.S. expressed as pyruvic acid in mg per 100 gm) in 108 samples of blood and 48 specimens of C.S.F.; that in the florid cases of beriberi the levels of these substances rise to four times the resting normal or 'cured' levels (in ten patients with the acute disease all but one of whom died) and that during recovery of the one patient the B.B.S. showed a progressive fall as the intensity of the symptoms subsided. We also reported that subacute beriberi cases, all of which recovered, showed only a small increase of B.B.S., which returned to normal level in a few days, the treatment usually being only rest in bed. Two cases of the atrophic type of beriberi had normal amounts of B.B.S., and a number of cases simulating clinically in some ways those with severe vitamin B_1 deficiency also showed no increase of B.B.S. Pyruvic acid was isolated from the blood of three different cases, also from the urine of one patient; it was identified by melting point determinations and by quantitative analysis (4). The presence of pyruvate in the cerebro-spinal fluid was reported in 1935.

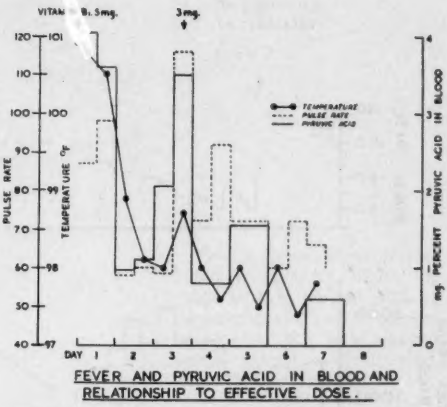


FIG. 4. Response of blood 'pyruvic acid' and pulse rate to administration of vitamin B_1; also associated rises in temperature, pulse rate and 'pyruvic acid,' necessitating further dosage with the vitamin.

Also, positive reactions had been noted for methyl glyoxal in blood and urine. These early findings are amplified and discussed elsewhere by Platt and Lu (4), Platt (20) and Platt and Lu (5).

In view of the recent work on the quantitative estimations of carbonyl compounds in blood urine and cerebrospinal fluid, I should say that the figures I shall give you for 'pyruvic acid' determined by the hydrazone method should be for 'carbonyl compounds' expressed as pyruvic acid. None of this new work seems to me to affect the validity of our main observations, and we fully recognized the possible significance in body fluids of other substances than pyruvic acid which either bound bisulphite or formed 2:4 dinitrophenyl hydrazones (5).

I want here to mention the possible value for clinical work of a simple nitroprusside reaction for pyruvic acid; this reaction was described by Simon and Piaux (21).

In figure 3 some recovery curves are reproduced—these show that vitamin B_1 very rapidly restores the level of blood pyruvate to normal.

The rapidity of this change reflects, in my opinion, the rapidity of the accumulation of the metabolites determined. The speed with which these changes, accompanied by circulatory and other manifestations of the acute disease, develop is well illustrated

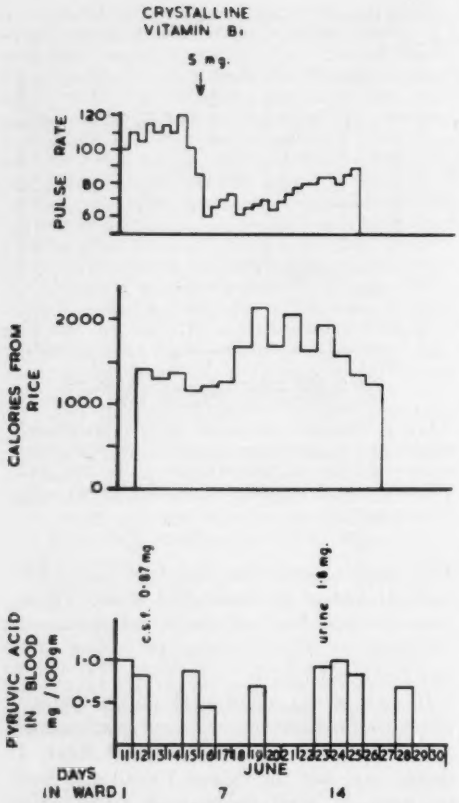


FIG. 5. Increased intake of food after treatment with vitamin B_1 of patient with subacute beriberi and rise of pulse rate and blood 'pyruvic acid' consequent on increased caloric intake.

by an 'accident' which I shall describe later in connection with the effect of physical work on 'pyruvic acid' levels in the blood. It is appropriate here, however, to mention some points in the natural history of various forms of beriberi. In Shanghai, the crop of acute cases came at the time of the sudden change of climate from a mild spring weather to a hot, humid and exhausting summer season akin to Washington at its worst. The men affected were young adults, sometimes the supervisors of apprentices in the small factories, superficially better nourished than their charges. They probably got a lion's share of the food, especially the rice. They would probably, at the end of a long day's work, sit in the crowded alley-ways between the factory buildings, fanning themselves to cool off before returning to their work benches or closely packed bunks in the little factory building, to try to sleep. I have suggested elsewhere (22) that the attacks of the bed-bug with which these living and working quarters were incredibly heavily infested probably finally precipitated the onset of the acute fulminating phase of an attack of beriberi. It was customary for the patient to be brought to the hospital during the night in some stage of the clinical picture described by de Langen (16). It is reasonable to suspect that the susceptibility to the acute attack of beriberi may be related to the process of adaptation to the summer weather. I must stress the influence of humidity as well as of temperature increases, both of which undoubtedly contribute to personal discomfort and interfere with rest and sleep and, therefore, with metabolic recuperation after physical activity. Consideration of accentuating factors and environmental conditions do help to an understanding of the emergence of the various forms of beriberi (7).

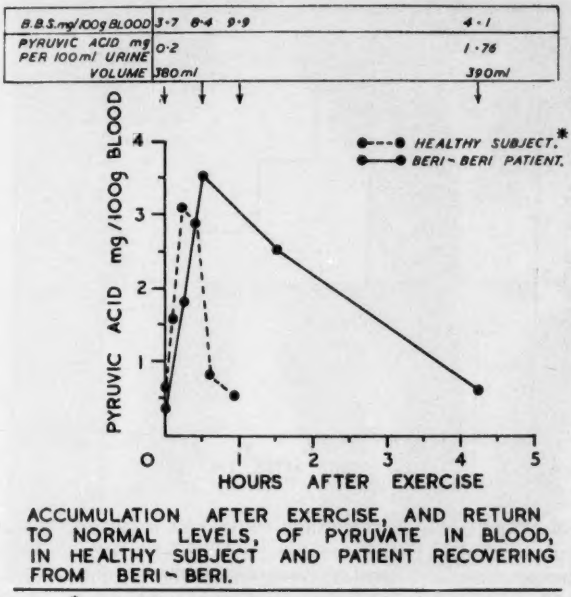
The frequent association of fever with the onset of subacute beriberi has already been mentioned. The effect of a rise in body temperature of the 'pyruvic acid' level in blood is shown in figure 4.

The patient with fever responds less readily to treatment with 5 mg vitamin B_1; recurrence of pyrexia in the third day led to a return of symptoms—tachycardia and a rise in blood pyruvic acid; these changes regressed when a further 3 mg of vitamins was administered.

One sign of vitamin B_1 deficiency, recognized in early research, is the loss of appetite. The calorie value of the food eaten by a patient with subacute beriberi before treatment is shown in figure 5.

After administration of vitamin B_1 the appetite improved, as shown by the increased caloric intake. This was provided mainly by highly milled cooked rice and the result of increased food consumption is seen from the figure—a slow rise in the pulse rate, an increase in the resting level of 'pyruvic acid' in the blood and an appreciable concentration of 'pyruvic acid' in the urine several days after the increased food consumption.

Exercise is known to increase the blood


B.B.S. mg/100g BLOOD 3·7 8·4 9·9 4·1
PYRUVIC ACID mg PER 100ml URINE 0·2 1·76
VOLUME 380ml 390ml
HEALTHY SUBJECT.*
BERI-BERI PATIENT.
PYRUVIC ACID mg/100g BLOOD
HOURS AFTER EXERCISE
0 1 2 3 4 5
1 2 3 4

ACCUMULATION AFTER EXERCISE, AND RETURN TO NORMAL LEVELS, OF PYRUVATE IN BLOOD, IN HEALTHY SUBJECT AND PATIENT RECOVERING FROM BERI-BERI.

* DATA FROM JOHNSON AND EDWARDS (1937)

FIG. 6. Increase in and delayed removal of blood 'pyruvic acid' of patient with hypovitaminosis B_1 after exercise; note high B.B.S. level and output of 'pyruvic acid' in urine.

pyruvic acid of the healthy subject to about the same level as that found in patients with acute beriberi (23). In the healthy subject after exercise, however, the level rapidly returns to a low value; in the thiamine-deficient subject, a high level is maintained and, in the curve shown in figure 6, it took about 4 hours to return to the resting level.

The concentration of 'pyruvic acid' in the urine passed at the end of the exercise was several times that of the urine at the beginning of the test. This subject failed to complete the usual stair-climbing test applied to patients about to be discharged from hospital. During the test, he complained of weakness of the legs, he was distressed and cyanosed, his pulse rate increased from 70 beats per min. at rest to 110 and his blood pressure altered from 110/80 per min. to 70/40 per min. at about 40 min. after the commencement of the climb. For a while he resembled in many respects a patient in the fulminatory stage of beriberi. This subject showed an increase in B.B.S. far in excess of the amount accounted for by the accumulation of 'pyruvic acid' (24).

The rapidity with which the biochemical changes in the blood are restored to normal parallels the speed with which the increase occurs; other disturbances may be slower to develop and correspondingly slow to regress. Thus diuresis in an edematous patient generally does not begin for 3 to 4 days after treatment as shown in figure 7.

The time taken for the dilated heart to return to normal takes several weeks. The series of teleradiographs reproduced in figure 8 (reading clockwise) were taken at intervals of 10 days.

In my experience, with one exception, patients with acute beriberi had tachycardia. In several instances, after treatment, and sometimes for days, the treated patient had bradycardia. Pulse rates as low as 40 beats per min. have been recorded; the low rate is temporarily restored to normal by an inhalation of amyl nitrate. Hawes *et al.* (15) were impressed with "the return of a palpable pulse in a collapsed pulseless patient and the gradual hardening or rather stiffening of the peripheral arteries that occurs from hour to hour until . . . they feel like peripheral arteries of an essential hypertension."

There are interesting responses to adrenaline—vascular and biochemical (some observations made with one of my patients have been written up by Lim in 1938, who

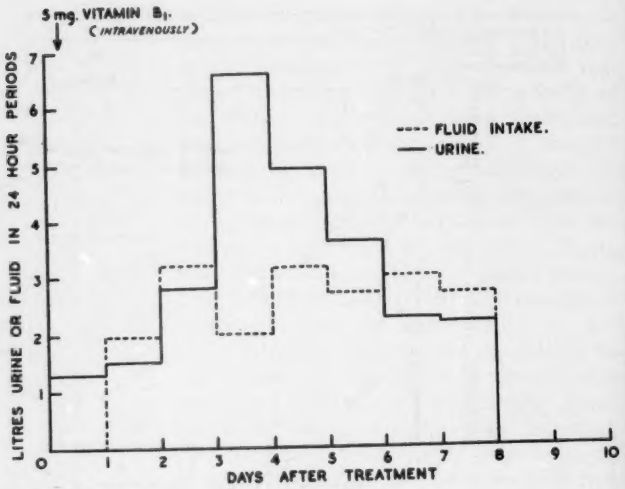


FIG. 7. Marked diuresis in beriberi patient starting 3 days after treatment with vitamin B_1.

also studied a further series of patients in Hong Kong). Some representative data showing the effect of adrenaline administration are set out in table 3. The vascular response to the administration of adrenaline is different in the treated as compared with that in the untreated patient. Signs similar to those seen in the acute phase of beriberi developed during the adrenaline test in the untreated patients; these include increased pressure in the veins of the neck, a diffuse pulsation in the precordial and epigastric regions, accentuation of heart sounds and murmurs, palpitation and a sense of profound discomfort.

The investigation of the effect of adrenaline in beriberi was not undertaken because of the work of Aalsmeer (25), of which we were aware, but because of the effect of the administration of adrenaline on the blood pyruvic acid recovery curve; an illustration of this effect is given in figure 9.

The report on this patient (2456/37) in the paper by Lim (9) is incorrect in that it is stated that the patient was initially given 2 mg of vitamin B_1 and 2 hours later he was given 5 mg; his interpretation of the course of recovery as inadequate dosage in the first place is incorrect. The interest of this patient to me is that 2 hours after admission, when he was given 5 mg of vitamin B_1, he stopped breathing. I gave 1 mg adrenaline by the intracardiac route and a further 0.5 mg 5 minutes later. With the application of artificial respiration the patient revived.

Further study was made of the effects of adrenaline on the blood chemistry; one example is given in figure 10. The results shown in figure 10 clearly demonstrate that the alterations in blood pyruvate and sugar produced by adrenaline are not so marked in the beriberi patient after vitamin B_1 has been administered.

It has been indicated above that recovery from acute beriberi is rapid; nevertheless, a little time is needed for phosphorylation of the thiamine and for the initiation of the restoration of normal biochemical mechanisms.

It is interesting to find that in one group of patients who died shortly after treatment was given, their blood sugar values were consistently low. Some results on a series of 18 patients are given in table 4. Not only is a fatal issue to the disease associated with low values for the blood sugar, but there is also a tendency for the B.B.S. to be relatively high.

I have tried in the foregoing presentation to confine myself to some highlights; it is inevitable that such a presentation cannot be complete and I am fully aware of numerous omissions, some of which can be filled in by consulting the literature, especially the papers referred to in the text. I single

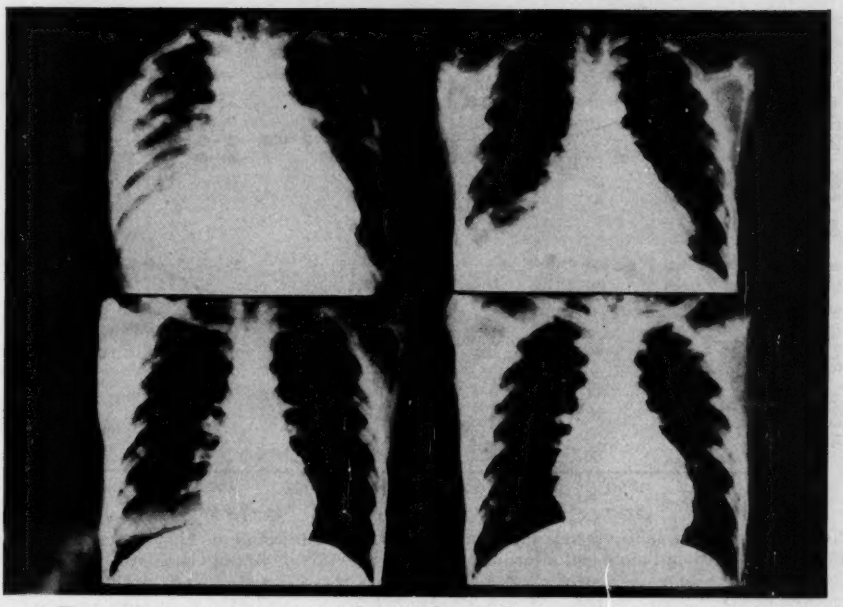


FIG. 8. Teleoradiograms taken at intervals of 10 days during treatment with vitamin B_1 of patient with beriberi—read clockwise.

TABLE 3. BLOOD PRESSURE CHANGES IN VITAMIN B_1 DEFICIENCY IN MAN AFTER ADMINISTRATION OF ADRENALINE SUBCUTANEOUSLY

Case No.	Degree of Defic.	Amt. of B_1, mg	Amt. of Adrenaline, mg	Blood Pressure: mm Hg at Intervals, in minutes																	
				Systolic						Diastolic						Final Point					
				Initial	5	10	20	30	40	Initial	5	10	20	30	40	Initial	5	10	20	30	40
2466/37	2+		1.2	100	94	96	92	98		48	48	48	44	42		20	10	0	0	0	0
		5	1.2	110	130	142	142	142	140	70	78	78	74	70	68	50	50	40	30	16	0
2410/37	3+		1	118	118	114	114	112		80	70	60	64	62		50	30	28	20	10	0
		5	1	134	162	138	134	132	132	96	94	80	84	82		88	80	64	64	64	64
1681/37	4+		1	122	128	124	128	132		78	60	48	38	36		48	10	0	0	0	0
		3	1	128	130	130	136	136		62	58	54	52	52		50	48	42	26	0	

out for special mention our observations and comments on methyl glyoxal (4, 5). I must also mention a question which has worried me for many years, i.e. did I miss Wernicke's encephalopathy and Korsakoff's psychosis in beriberi in Shanghai? According to Victor (26) the crux of the clinical picture is in the ocular disturbances. Of the signs he mentions I have to say that in all the cases of beriberi I have seen—several hundreds—only in two cases have I seen bilateral nystagmus.

In my view we must extend our investigations in thiamine deficiency, especially in the florid form, to include the pathology of the endocrine glands, notably those concerned in the regulation of carbohydrate metabolism. Also, in view of the likelihood that in case 2456/37 the respiratory failure was due to a biochemical lesion in the central nervous system, more attention should be paid to the possible crucial role in the terminal events of the acute disease (notably in infantile beriberi) to the central nervous system. "... the failure in energy supply at the pyruvate step must distort the metabolism of the brain cells and throw the nervous system out of gear" (27).

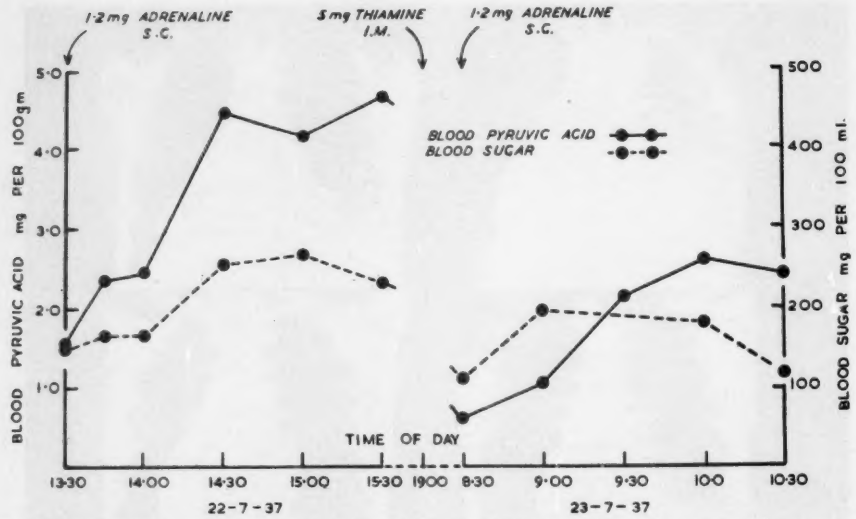

1·2 mg ADRENALINE S.C.
5 mg THIAMINE I.M.
1·2 mg ADRENALINE S.C.
BLOOD PYRUVIC ACID
BLOOD SUGAR
BLOOD PYRUVIC ACID mg PER 100 gm
BLOOD SUGAR mg PER 100 ml.
TIME OF DAY
13·30 14·00 14·30 15·00 15·30 19·00 8·30 9·00 9·30 10·0 10·30
22-7-37
23-7-37

FIG. 9. Response to subcutaneous injections of 1.2 mg of adrenaline of blood sugar and 'pyruvic acid' in beriberi patient before and after intramuscular injection of vitamin B_1.

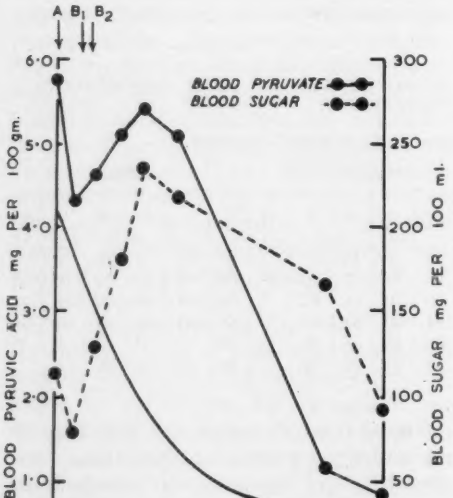


FIG. 10. Changes in 'pyruvic acid' and sugar (Folin and Wu method) in blood following treatment of patient with fulminating beriberi with vitamin B_1; respirations ceased in 1 hr. when 1 mg and 0.5 mg adrenaline given by intracardiac route together with artificial respiration. Patient completely recovered. Smoothed curve from fig. 2 shown for comparison.

At the conclusion of this effort to rake over the past, in excuse for the imperfections of which I must plead not only the lapse of time but also the loss of some pertinent records, I want to confess to a strong feeling of indebtedness to the organizers of this meeting for the stimulus it has been to me. I had always promised myself that I would return to research on beriberi, and, indeed, we have already some work in progress on the effect of thiamine deficiency in the mother on her young. Moreover, as will be apparent from recent publications (22, 28, 29), we are actively engaged in research into the effects of high-carbohydrate–low-protein diets in animals, and there are some obvious inter-relationships between this work and several facets of the beriberi problem.

HUNDLEY: Would Platt comment on the mechanism of the edema in beriberi?

PLATT: No. I think we might get clues from the work on the heart muscle and the changes that go on in the ECG. We produced comparable changes to those that you get in beriberi in high-carbohydrate–low-protein animals just recently.

MCLAREN: I'd like to mention the optic nerve lesions in association with beriberi. Early Japanese workers laid great emphasis on these. Most clinical experience leads us

TABLE 4. VALUES FOR PYRUVIC ACID AND BISULPHITE-BINDING SUBSTANCES IN BLOOD AND CEREBROSPINAL FLUID OF PATIENTS WITH LOW, NORMAL AND HIGH VALUES FOR BLOOD SUGAR

Case No.	Blood		C.S.F.		Blood Sugar	Comment
	P.A.	B.B.S.	P.A.	B.B.S.		
	mg/100 gm	*mg/100 gm*	*mg/100 gm*	*mg/100 gm*	*mg/100 gm*	
1	1.7	11.2	1.0	4.2	32	died with hypoglycemic convulsions
	3.4	11.7	1.7	3.7	60	
2	1.2	12.3	1.1	3.8	64	died
3	2.6	16.3	1.9	9.2	65	died
4	5.5	11.2			66	died
5	4.1	18.3	3.3	9.0	68	died
	4.3	19.6				—values 90 minutes later
6	1.2	5.8			78	
7	3.3	7.5	1.9	4.6	82	died
8	3.5	9.5			84	
9	4.6	11.2			88	
10	1.7	5.8	0.9	4.6	105	
11	2.3	6.7	1.1	6.4	111	
12	3.2				112	
13	5.8	13.2	2.9	7.2	114	
14	1.9	7.2			117	
15	4.1	10.2	2.8	6.4	146	fever
16	1.8	12.7	1.6	6.8	154	
17	2.4	7.7	2.2	3.4	164	died + fever
18	1.2	21.4	1.0	5.6	192	died + pneumonia

to believe that thiamine, of all the B complex vitamins, does have the greatest effect in reversing the early optic nerve lesions, although best results have usually been obtained with the total B complex. I think, too, that this would be a very useful indication in field work. I'd like to know whether ophthalmoscopic examination is part of the clinical surveys which have been done.

HIGGINSON: The thing that has really confused me has been the reports of Occidental workers who have referred to nonthiamine or thiamine-resistant disease and made the diagnosis of beriberi heart disease in recent years, especially in the American literature; quite frequently in cases on which the evidence of thiamine deficiency in my opinion is both illogical and nonexistent.

FOLLIS: How much salt is in the diet of the native rice-eater who develops edema?

PLATT: The Chinese eat a considerable amount of salt in soy sauce, which is used daily. I mentioned that in Lim's observations on the development of chronic beriberi he kept his patient on a diet of salted vegetables and salted egg. Salt is used a great deal for preserving and in cooking, so there's quite a bit of sodium chloride in the diet of the Chinese.

BURGESS: May I ask Ramalingaswami to talk on this, because I know he's done quite a bit of work on salt intake.

RAMALINGASWAMI: Before salt, I should like to comment on the question raised by McLaren about optic nerve lesions; there is some work done by Rodgers (121) on experimental thiamine deficiency in the rat, where he demonstrated degenerative changes in the optic nerve. He also went on to induce double deficiency of thiamine and riboflavin and found even more accentuated nerve damage.

I should like to ask Platt about the relationship between the accumulation of pyruvate and other intermediate compounds and the pathological manifestations in beriberi. I presume this question will come up again later. The salt intake in South India is something on the order of about 18.5 gm per head per day. This is perhaps the highest in India because as one goes up North the salt intake is somewhat less.

PLATT: The best answer I can give has

already been printed in a paper in the *British Medical Bulletin* (20). I've got nothing else to add.

PATWARDHAN: I take the liberty of being rather skeptical about the utility of pyruvate determination in blood for field work, as has been suggested by Platt. Our experience shows that such determinations have not given us the indication of the susceptibility of the population to beriberi in the areas where the disease is supposed to be endemic. In view of the present unsatisfactory situation, field workers interested in the studies on beriberi would like to know what are the things they ought to look for apart from the actual clinical signs and symptoms in order to be able to evaluate the state of vitamin B_1 deficiency in a population group.

III. The Pathology of Endemic Beriberi

FOLLIS: From the clinical aspects of beriberi which Platt has just told us about, one might expect to find rather pronounced changes in the nervous tissues and in the heart at autopsy. Most of our knowledge of the pathologic anatomy of beriberi is based on studies which were made by Japanese, Dutch and a few German pathologists at the turn of the century. Virtually no data have been reported since that time, so that our present knowledge is hampered by a great lack of information. The changes which were described in the peripheral nerves gave rise to the idea that beriberi was characterized by a peripheral polyneuritis. The lesions consist of a loss of myelin and a disruption of the axoplasm of nerve fibers. I wish to point out that at the time these studies were made beriberi was not looked upon as a deficiency disease. It was thought to be an intoxication, perhaps related to arsenic, oxalate, carbon dioxide poisoning or perhaps to food poisoning; another group thought that it was due to some infection, whether by animal parasites, bacteria or fungi. Therefore, the pathologists who studied cases of beriberi at autopsy may have been somewhat biased as to what they thought they ought to see in relation to etiology. They demonstrated changes in the neurone itself, in the sensory ganglia, and actually some pathologists, for instance Dürck (30), pictured degeneration in the spinal cord of patients who had chronic beriberi. It's important that no involvement of the brain was noted by these investigators. Only occasional changes were found in neurones of the medulla. We can summarize by saying that we just don't have very much information concerning the pathologic anatomy of the nervous tissues at a time when beriberi was so prevalent. There has been none added since (31).

Now we must mention another syndrome which Denny-Brown will discuss more in detail this afternoon; this is Wernicke's disease, which was described in the latter part of the last century as a clinical triad consisting of clouding of consciousness, ophthalmoplegia and ataxia. Rather characteristic changes were found in the brain at autopsy. Such consisted of symmetrical areas of necrosis and hemorrhage, particularly in areas such as the mammillary bodies and the nuclei about the third ventricle. I think it's important to point out that beriberi and thiamine deficiency are not synonymous terms. Moreover, Platt indicated he had never observed this syndrone in areas where endemic beriberi was occurring. It has mainly been seen in this country and in Europe, associated in part with alcoholism and, of course, with other forms of chronic malnutrition.

The other area of interest is the heart. Data concerning the morphologic changes which have been described in the heart in cases of beriberi coming to autopsy in the Far East are not very satisfactory. Dilatation of the heart, sometimes with hypertrophy, was described (31). Cloudy swelling, fatty infiltration and fragmentation of the myocardial fibers were described. Foci of necrosis were also pictured. The changes which have been described more recently in studies which were made in this country on the Occidental type of beriberi have stressed edema of the heart muscle and hydropic change in the heart muscle fiber itself. Virtually no alterations of an inflammatory nature or severe foci of necrosis have been found. We are faced, then, with the problem

of inadequate descriptive material on classical beriberi, both with respect to the central nervous system and to changes in the heart. Hence, modern autopsy studies are greatly needed.

HIGGINSON: I will briefly review some of the peculiar forms of heart disease that have been reported from the African continent. In Dakar, a form of heart failure known as 'thrombotic surgical heart' has been described (32). In this condition patients die suddenly a few hours after a surgical operation. At autopsy the heart is dilated and ante-mortem thrombi are found in the ventricles and atria. The etiology is unknown, though various hypotheses have been suggested including a possible relationship to malnutrition.

A group of unusual cases of heart failure in West African troops was briefly described by Bedford and Konstam (33). At autopsy endomyocardial necrosis associated with intracardiac thrombi was said to be present. This report has never been amplified. Later, Davies reported a form of heart disease common in Uganda which he called 'endomyocardial necrosis' (34). In this condition there is severe endocardial fibrosis associated with myocardial degeneration especially in the subendocardial region. Intracardiac thrombi are also present. The cause of the condition, which appears very similar to that seen in West African troops, is unknown but Davies and his colleagues do not believe that it is due to malnutrition (35). There is some doubt in my mind as to whether the fibrosis may not mainly represent organization of intracardiac thrombi.

In 1950 Gillanders in South Africa reported a series of cases of heart failure in the Bantu which he called 'nutritional heart disease' (36). His patients were admitted to hospital in severe cardiac failure, with tachycardia, and gallop rhythm. The cardiac output was low (1–2 l/min.); the heart appeared weakly pulsatile on x-ray. Many of these patients showed a mild peripheral neuropathy. Following treatment with a good ward diet, bed rest, digitalis and mercurials, the heart decreased in size and the patient often left hospital apparently recovered. On returning home, however, relapse frequently occurred and they were readmitted to hospital, each time in more intractable failure, which eventually led to death.

The pathologic picture was essentially that of a dilated and hypertrophied heart (37) which frequently contained intraventricular thrombi. Intracellular edema and 'hydropic' degeneration of the muscle were described, also focal myocardial fibrosis and degeneration. We have tended to regard these myocardial lesions as nonspecific, but sometimes they were clearly secondary to an overlying endocarditis. It appeared that the basic pathogenesis was a failure of the myocardium leading to cardiac dilation and hypertrophy, followed by secondary intracardiac thrombosis.

A very similar type of heart condition is found in Rhodesia, but failure is said to be irreversible (38). In one heart forwarded to me by Dr. M. Gelfand of Salisbury I could find no obvious abnormality.

The clinical type of idiopathic heart disease seen in Africa is a real problem, and in Johannesburg forms approximately one-third of cardiac admissions. Gillanders regarded the form of heart disease described by him as a manifestation of chronic malnutrition (not under-nutrition) and described recurrence of failure in a few patients who are given a poor diet after initial recovery. This experiment has never been repeated, but the nutritional origin of these cases has been opposed by others. The pathological evidence, however, supports some form of metabolic myocardial disfunction. It is of interest here to note that Grusin (41) has found a high frequency of EKG abnormalities, indication of myocardial damage in Bantu patients and healthy nurses with no evidences of heart disease.

Finally, Griffith (42) has recently demonstrated that several forms of acute reversibe heart disease occur in the South African Bantu. Some of the cases are identical with cases of Oriental beriberi and respond well to thiamine administration. Others do not respond specifically to thiamine. The etiology of the latter and their relationship to the chronic form described above is unknown.

It is probable that many of the cases represent some forms of myocardial inadequacy among the peoples of Africa, the etiology of

which is unknown, but some appear related to malnutrition. The pathological and clinical picture is similar to that described in what is called 'Occidental beriberi' (43). The nature of the latter condition is still far from clear, but not all such cases are beriberi.

KINNEY: Recently we studied an enlarged heart from a 55-year-old man who had been in congestive failure and died from a large pulmonary embolism. The lesions on the endocardium were similar to those just described. In addition, there were several thrombi in both auricles and in the right ventricle. There were no significant changes in the myocardium, valves or coronary arteries. We reviewed material from about 1,500 autopsies and found 5 additional cases with this type of endocardial lesion. One of these cases, also died with congestive failure. In the other cases, death was not associated with heart disease. In none of the 6 cases was nutrition considered a factor. Similar cases have been reported from other hospitals in this country. This suggests that at least some of the endocardial lesions represent a nonspecific reaction to etiological agents other than nutritional factors.

TEJADA: I want to present some material from Latin America which may have some resemblance to the cases from Africa described by Higginson.

This condition is mainly characterized by marked hypertrophy and dilatation of the entire heart without any evidence of other cardiac disease. On the surface of the epicardium one often observes 'milky spots,' characterized histologically by subepicardial inflammation or fibrosis. The myocardium usually is pale and in some cases shows irregular areas of diffuse or focal fibrosi. Sometimes the myocardium at the apex of the left ventricle presents diffuse fibrosis throughout its entire wall so that the ventricle protrudes out into the pericardial cavity or as an aneurysm at the apex of the left ventricle. Frequently there is evidence of mural thrombosis. This disease produces embolic phenomena in practically all the organs and, as a matter of fact, many of the patients come to the hospital complaining of these secondary effects.

On histological examination, there is evidence of a diffuse interstitial myocarditis. The cellular infiltration is formed by mononuclear cells as well as polymorphonuclear leucocytes, and some eosinophiles. There is a progressive transformation of this inflammatory reaction into fibrous tissue, so that finally one finds diffuse areas of severe fibrosis, sometimes so large that they can be seen grossly and resemble myocardial infarcts. In no cases have valvular or vascular lesions been observed. The etiology and pathogenesis of this condition is still not known. Brazilian investigators believe that is the chronic stage of Chagas disease. Several investigators in Venezuela considered that this condition has something to do with malnutrition and parasitic infestation. Jaffe (44) in Venezuela thinks that there is some allergic reaction related to several factors, among them vitamin B_1 deficiency. I don't know what the etiology may be. Probably, it has something to do with Chagas disease, although I have not been able to see any parasites even though in many of my cases I have studied up to 100 histological sections from each heart.

SCRIMSHAW: I would like to mention briefly that despite the many other serious nutrition problems of Latin America, beriberi is not a health problem. Not only is the scientific literature from Latin America lacking in reports of the occurrence of beriberi, but also dietary and clinical surveys give no indication that thiamine deficiency is of appreciable significance. INCAP studies in Guatemala of the group most likely to be deficient in thiamine, the pre-school children, have indicated that even in low-income rural groups when deficiencies of protein, vitamin A and riboflavin were all pronounced, thiamine intake averaged .64 mg per day and urinary excretion 148 μg (S.E. 29) per 24-hour sample. In this group thiamine excretion per gram of urinary creatinine was 1,055 μg (S.E. 242) as compared with 1,397 (S.E. 381) for well-nourished urban children consuming twice as much thiamine. Thus when calculated on the basis of creatinine, the much smaller muscle mass of the low-income rural preschool children offset their lower thiamine intake and smaller total excretion.

Even in acute kwashiorkor in Central America, serum thiamine values average 4.33 mg per 100 ml (S.D. 19). This average increases to 6.88 (S.D. 1.03) during treat-

ment but both averages are within normal limits. While these finding are most representative of the populations in Mexico, Central America, Venezuela, Columbia, Ecuador, Peru, etc., where corn (maize) is the basic staple of the diet, there is no suggestion of thiamine deficiency among the people of Panama, and some coastal areas of South America where rice replaces corn as the principal cereal. In these latter populations it never forms as large a proportion of the total diet as in the Orient.

UEHLINGER: At the end of the Second World War, I had the opportunity to observe some prisoners released from German concentration camps. The time of undernutrition varied between several months and 2 years. The daily caloric intake in the last months was about 500–800. The autopsy findings in 50 cases were as follows: total loss of subcutaneous tissue, wasting of the skeletal muscles, diminution of the heart weight in relationship to the fall of the body weight (1/200). In no case was there a marked dilatation of the right ventricle or of the right auricle. Histologically the myocardium showed a marked degree of brown atrophy, but no fatty degeneration or fibrosis. The coronary arteries showed varying degrees of sclerosis. There was no case of clear-cut vitamin B deficiency.

FOLLIS: In relation to what Higginson said about Occidental beriberi heart disease, it should be pointed out that the American Heart Association lists eight criteria for the diagnosis of beriberi heart disease. The sixth of these is 'lack of other recognized cause of heart failure'. Hence, the term 'beriberi heart disease' in this country has been used as a catch-all for any type of undiagnosed heart disease.

IV. EXPERIMENTAL THIAMINE DEFICIENCY

FOLLIS: We'd now like to say something about experimental thiamine deficiency. As you all know, in the late 30's and early 40's, after most of the crystalline vitamins became available, a number of studies were reported on the effects of what was probably uncomplicated thiamine deficiency in various species: rats, dogs, foxes, swine, monkeys (31). Certain physiological changes, such as bradycardia, alterations in the electrocardiogram, decreases in thiamine or cocarboxylase content of the myocardium and other tissues, were described. So, too, changes in the oxygen quotient of myocardium and an increase in pyruvate content of the blood were described. The lesions, as far as the heart is concerned, consist of a widespread foci of necrosis with a rather pronounced inflammatory reaction. The cell fibers die, leukocytes come in and are replaced by connective tissue fibers. Such animals may have become cyanotic, short of breath, die suddenly, and show profound, though not specific, electrocardiographic alterations. I think there can be no doubt that thiamine deficiency in the presence of all other known nutrients may lead to physiological and anatomical changes in the heart (31).

It is of interest that the skeletal muscles do not show alterations as a result of thiamine deficiency nor have changes been seen in smooth muscle.

We come to a very complicated situation when we consider thiamine deficiency and the nervous system. In all of the studies that have been made of the peripheral nerves of experimental animals deficient in thiamine, no alterations have been described that could not be accounted for by simple inanition (31). There is a recent report by North and Sinclair (43) that may negate this statement, but if so it is the first demonstration that in rats chronic thiamine deficiency may lead to changes in the peripheral nerves. The only compelling evidence, of course, for the relationship of thiamine to the integrity of the peripheral nerves stems from the studies in birds, which began with Eijkman. In Aves degeneration of myelin and axoplasm is seen as a result of thiamine-deficient diet; this apparently has no relation to inanition.

It is of interest that lesions of the central nervous system have been seen in a number of mammalian species: rats, cats, swine and monkeys. These resemble, with certain exceptions which are not of very great magnitude, the changes which are seen in Wernicke's disease, as it occurs in man (31).

There have also been studies of experi-

mental thiamine deficiency in man. In such individuals changes of a rather minor character have been noted in the electrocardiogram. A non-specified 'neuritis' has been described and, in association with this, edema, anorexia, vomiting, as well as certain neurological and muscular changes, such as weakness, ataxia, etc., have been described (46, 47).

Now then, I'd like to discuss one other type of experiment. We now have a baseline as far as the effects of most of the essential nutrients are concerned, so that it has seemed as though it might be worthwhile to begin to study, experimentally, multiple deficiency syndromes. During the last several years we have begun such observations in our own laboratory. A group of African monkeys have been placed on a diet of polished rice unenriched except for vitamin C, because the animals will develop scurvy and die if ascorbic acid is not given to them. We have been fortunate to produce a rather interesting and dramatic alteration in the heart muscle. For instance, after one young growing animal had been on rice for about 7 or 8 weeks he became prostrate early one afternoon and was dead that evening. When you touched him before death he wasn't able to cry out. He lay on his side in obvious respiratory distress. At autopsy, grossly, there were no changes and it wasn't until microscopic sections were studied that we found profound alterations in the myocardium. There was necrosis of the myocardial fibers, without any inflammatory reaction. One finds that some fibers are swollen and have lost their striations while others have completely disintegrated and are undergoing a sort of lysis. We have seen this now in 4 animals. In one instance the monkey had been on rice for about a year and seemed to be showing nothing save that his growth had plateaued. When we added 10% casein to the rice diet, within 10 days he was dead, succumbing overnight. At autopsy the next morning fresh necroses were found in his myocardium. This observation brings out something which we are going to come back to when we discuss pathogenesis: the very great importance of the inter-relations of various nutrients in producing pathologic changes of one type or another and the importance of the general level of metabolism on the development of these lesions. Clinically, we have seen no evidence of any neurological effects of a rice diet in the monkey, nor have we found any changes at autopsy either in the brain or in the peripheral nerves. Hence, we have been disappointed in attempts to reproduce the neurological aspects of beriberi; our studies on the cardiac changes produced by polished rice are continuing.

OLSON: For some time we have been interested in the biochemical pathogenesis of cardiac failure of different types. In view of some of the discussion this morning which has questioned the reality of cardiac failure in beriberi, I should like to present some data relevant to this question. Part is derived from the study of experimental animals and part is obtained from the study of clinical material.

We began this general program of research by studying the *in vitro* metabolism of cardiac muscle obtained from rats and ducks fed thiamine-deficient diets. Under these conditions we were able to show (48) that the rate of pyruvate utilization by slices of thiamine-deficient rat and duck ventricle was significantly reduced. Further, there was a relationship between the rate of pyruvate disappearance and thiamine content of the ventricle. In figure 12 is shown a plot of pyruvate utilization expressed as a metabolic quotient (pyruvate disappearing per mg dry weight per hour) against tissue cocarboxylase expressed in terms of micrograms of thiamine per gram of fresh duckling heart muscle. It can be seen that the pyruvate utilization did not fall in these slices until the thiamine content was reduced from a normal value of about 10 μg per gm to about 2.5 μg per gm. Further reduction in thiamine content resulted in a precipitous drop in pyruvate utilization. The concept that there are no reserves of the water-soluble vitamins is erroneous, although specific B-complex nutrients vary widely, as evidenced by the deviation of the 'incubation periods' for various B-complex deficiency syndromes.

The next question was to determine whether the intact heart behaved like the cardiac muscle slice in thiamine deficiency. In table 5 are presented some representative data from a study by Hackel and Goodale

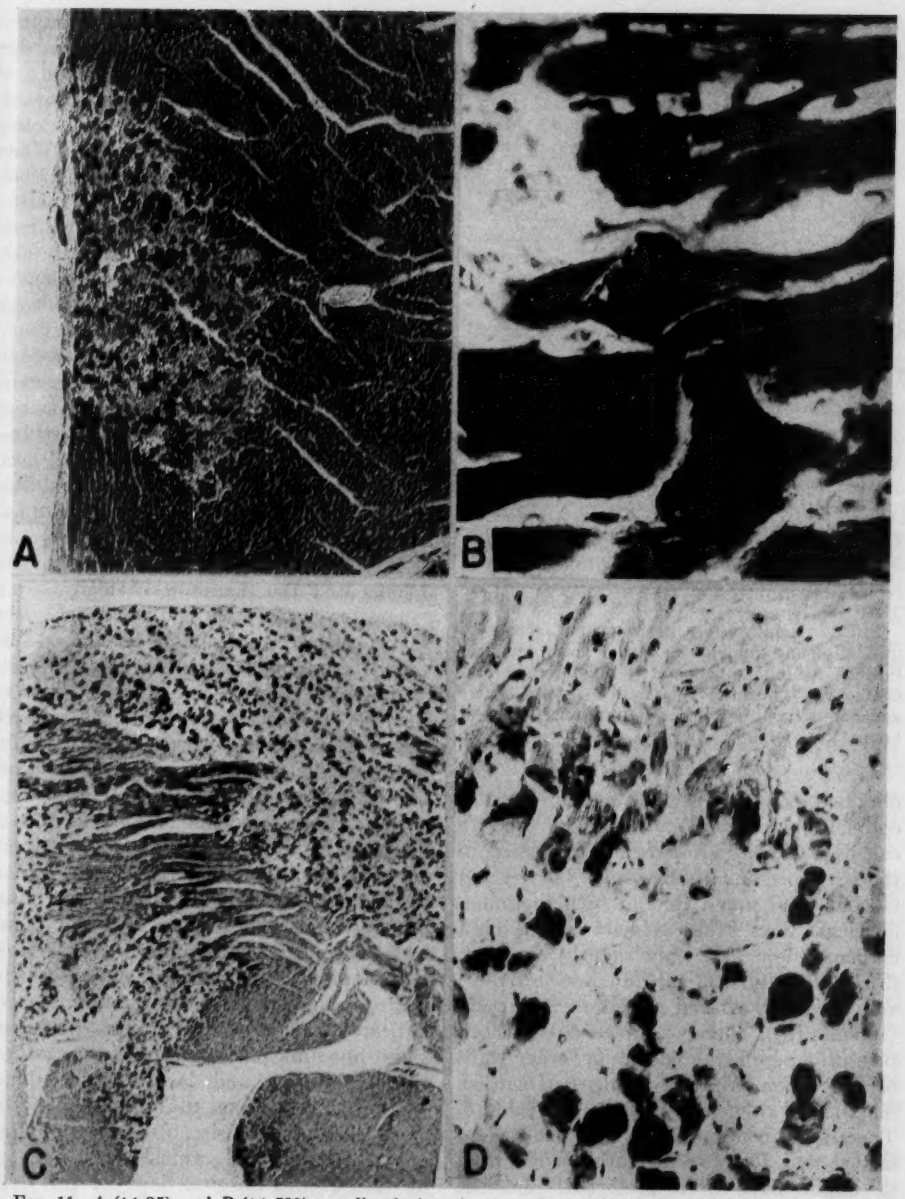


FIG. 11. *A* (× 35) and *B* (× 500), cardiac lesions in a monkey on diet of polished rice for 7 weeks. *C* (× 35) and *D* (× 265). Lesions in animals on diet on rice, supplemented with ascorbic acid for 35 weeks. Casein (10%) was then added; the animal died suddenly 3 weeks later.

(49) in which thiamine deficiency was produced in dogs by feeding an autoclaved diet. After signs of deficiency occurred, the metabolism was studied by coronary sinus catheterization. This technique enabled one to compare venous blood obtained principally from the left ventricle with arterial blood. These data, together with those

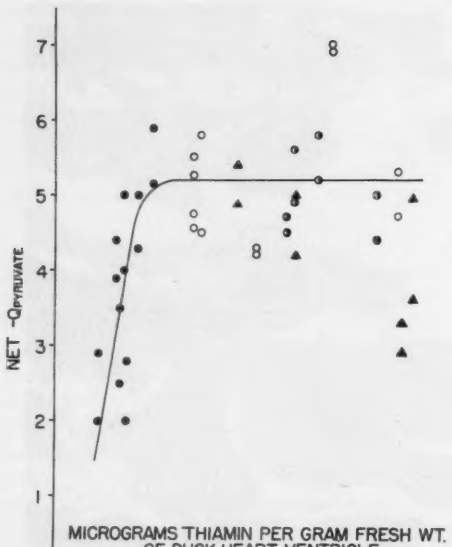


FIG. 12. Relationship between net pyruvate utilization and thiamine content of heart ventricle in ducks: thiamine-deficient (●), pen-fed normal controls (○), normal controls fed *ad libitum* (◑) and normal controls fed *ad libitum* and fasted 48 hours (▲). See (48).

obtained by an independent measurement of coronary blood flow, can then be used to calculate the oxygen and substrate usage of the heart per unit fresh weight. Hackel and Goodale (49) found that in such thiamine-deficient dogs, the cardiac output and coronary flow were only slightly elevated, (3.1 l/min. and 139 ml/min., respectively) and the mean arterial pressure was normal (106 mm Hg). There was no elevated venous pressure, peripheral edema or other signs of congestion. Marked changes in cardiac metabolism occurred, as shown in table 5. The arterial concentrations of pyruvate and lactate were found to be markedly elevated, as might have been predicted, and the extractions were markedly reduced in proportion to arterial concentration. The total contribution of carbohydrate (glucose lactate and pyruvate) to the energy needs of the heart averaged only 29% of the total, suggesting that despite elevated levels of these substrates the heart was depending chiefly on fatty acids for energy production. Further, we can conclude from these experiments and similar ones carried on in our own laboratory that despite the absence of signs of congestion, the heart was participating in the generalized metabolic defect induced by thiamine deficiency. When congestive heart failure does occur in association with beriberi, it is the result of the circulatory stress (large venous return) produced by peripheral vasodilatation. In fact, we have produced transient congestion in thiamine-deficient dogs by infusion of large amounts of saline.

Although wet beriberi heart failure is comparatively rare in the U.S.A., occasional cases are seen (50). I should like to present such a case of a young 28-year-old schizophrenic white male alcoholic who had been drinking large amounts of whiskey and beer and eating poorly for 2 months prior to admission to one of the Veterans' hospitals in Pittsburgh. The admission findings fully documented the diagnosis of high output congestive heart failure. The weight was 75 kg; blood pressure, 124/50; pulse, 124; venous pressure, 235 mm saline. Electrocardiogram showed only minor T-wave changes. The heart was enlarged to both right and left and showed a typical water-bottle configuration on x-ray. There were fine crackling rales at the lung bases and both pulmonary edema and effusion were noted by x-ray. The liver edge was 8 cm below the right costal margin. On admission the blood glucose was 61 mg %; the blood pyruvate was 2.3 mg % and the blood lactate was 15.5 mg %. Figure 13 represents the hospital course of this patient. With bed rest, house diet and the vitamin therapy indicated, rapid improvement occurred. Pulse pressure, pulse rate, venous pressure, blood pyruvate and lactate came down promptly and edema disappeared. On the third hospital day a sizeable diuresis of some 10 kg was initiated, which was completed on the seventh hospital day. By the sixth day the circulation time had lengthened to 13 seconds, in harmony with the view that the cardiac output had decreased. The chest film showed appreciable decrease in heart size and loss of pulmonary edema on the fifth day and was essentially negative by the thirteenth. This patient gained lean body weight with continued good nutrition

and was discharged 'well' on the thirtieth hospital day.

Although we did not have an opportunity to study the cardiac metabolism of this patient, I had studied a similar patient with Goodale at the Peter Bent Brigham Hospital some years ago. This was a 30-year-old alcoholic with classical findings of high output cardiac failure: flushed hot skin; dyspnea, massive edema, B.P. 140/20, pulse 105, arm-tongue circulation time of 8 seconds (decholin), enlarged water-bottle heart and pulmonary congestion by x-ray. Cardiac catheterization revealed a cardiac output of 25 l/min. (cardiac index 10.3 l/m²/min.), coronary flow of 213 cc/100 gm/min., and elevated filling pressures for both ventricles. The cardiac respiratory quotient was 0.70. Although the blood pyruvate of this man was not particularly elevated, the extraction of pyruvate and lactate by his heart muscle was reduced below normal, as shown in table 6. It is becoming clear that marked elevation of fasting blood pyruvate is not a *sine qua non* in the diagnosis of beriberi. The infusion of glucose in this patient, however, resulted in abnormal elevations of pyruvate and lactate with only small changes in extraction of these substrates by the heart and with no change in respiratory quotient. This confirmed the defect in the terminal oxidation of carbohydrate by this patient's heart muscle and strengthened the diagnosis of beriberi heart disease.

In conclusion, these data support the view that in beriberi the heart participates in the general metabolic defect which characterizes thiamine-deficient tissues. Heart failure usually does not occur unless there is peripheral vasodilation, mediated either locally or centrally, with augmentation of venous return to the point where the heart cannot accommodate a further influx. At this point 'high output failure' ensues with sodium retention. Whereas the cardiac output is 'high', it is not as high as is needed to accommodate the venous return (51). It follows, I believe, that the occurrence of cardiac failure in beriberi will be variable, depending upon the extent of the severity of depletion of cardiac cocarboxylase and the extent of peripheral vasodilation. When it does occur, however, it accounts for some of the edema seen in both endemic and sporadic cases.

TABLE 5. CARDIAC METABOLISM IN THIAMINE-DEFICIENT DOGS (49)

Substrate	Content Mg. % or Ml. %		Extraction A-V	Oxygen Extraction Equivalent %
	Femoral Artery	Coronary Sinus		
Oxygen	18.9	5.6	13.3	
Pyruvate	5.2	4.1	1.1	6
Lactate	49.9	47.8	2.1	12
Glucose	71.0	69.5	1.5	11
Total				29

HANDLER: What per cent of the oxygen consumption of the heart is due to extracted carbohydrate, pyruvate and lactate?

OLSON: This varies widely, depending upon whether the individual is fed or fasted. Ordinarily in the postprandial human and dog about 80 to 90% of the fuel burned by the heart is derived from carbohydrates, and in the fasted human or dog about 30% is derived from carbohydrate and about 70% from fatty acids. These fatty acids, we now know, are derived from the so-called NEFA or nonesterified fatty acid fraction. One interesting contrast in that connection is that diabetics behave somewhat similarly to patients with thiamine deficiency as far as the overall pattern of carbohydrate metabolism of the heart is concerned. Diabetic cardiac muscle burns very little carbohydrate and mostly fat, yet cardiac failure in association with diabetes uncomplicated by coronary artery disease is a rarity. I wonder if the difference might not be that in beriberi vasodilation occurs, whereas in diabetes it does not. The total dependence of the heart upon fat as a source of energy may not be appropriate for high work loads. It is, of course, possible that thiamine deficiency may influence some other phase of cardiac metabolism.

HUNDLEY: We became interested several years ago in the dissimilarities between experimental thiamine deficiency in animals and Occidental or Oriental beriberi (52–55). These differences were not only in the clinical syndromes but also in pathology and electrocardiographic findings. A young growing rat fed a thiamine-free diet will die after 3 to 6 weeks with no specific clinical symptoms, and with no pathology in the

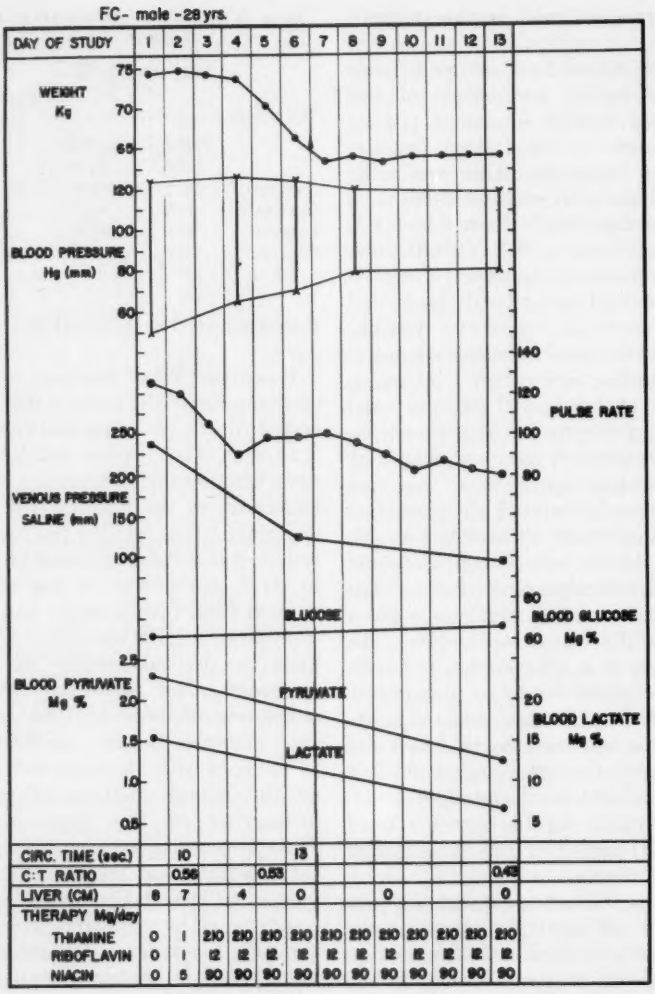


FIG. 13. Study of beriberi heart failure in an alcoholic.

heart, the central nervous system, spinal cord or peripheral nerves. It dies apparently from respiratory failure. If the thiamine deficiency is made more chronic, say 8 or 12 weeks, then something quite different develops. Clinically the rats develop a very characteristic syndrome with spasticity of the muscles, ataxia, convulsive seizures, and die in a convulsive state unless treated promptly. Pathologically, one finds very characteristic lesions in the heart that Follis described earlier. Lesions of this type have been reported rarely in human beriberi and never so severely as found in the rat. Similarly one finds very characteristic and very marked defects in the rat's electrocardiogram. These defects are mainly in the conduction system. Again, defects of this type are not characteristic of human beriberi. Under such conditions, the central nervous system, spinal cord and peripheral nerves show no lesions to ordinary pathological examinations. If one feeds young rats a polished rice diet one sees something still different. These diets were fortified in all known required nutrients except thiamine,

but of course contained the small amount of thiamine found naturally in polished rice (56). Under these conditions the deficiency develops slowly, but these animals die eventually with acute convulsive seizures typical of thiamine deficiency. However, no abnormalities are found either electrographically or pathologically. These facts suggest that the various characteristic lesions and manifestations of thiamine deficiency can be dissociated according to the acuteness of the deficiency and depending on whether the diet contains small amounts of thiamine. These facts also suggest that the relatively mild and relatively nonspecific heart lesions and electrocardiographic changes found in typical human beriberi may be due to the small amounts of thiamine provided by polished rice.

MACKENZIE: I'd like to ask Follis if the heart damage in his monkeys could account for their death, or does he attribute this sudden death to some other cause?

FOLLIS: The changes in the heart are extensive morphologically. Moreover, one can't say how much of a biochemical lesion may be present as far as the myocardial fibers are concerned. All this accounts for their exitus.

PLATT: I would like to ask if anybody knows whether there are any differences in the intermediate metabolism of alcohol and glucose which might account for some of the differences being found between Occidental beriberi, where often it's related to alcoholism, and Oriental beriberi when alcohol consumption is not effective in etiology?

PATWARDHAN: Follis mentions feeding his monkeys on polished rice alone with a certain amount of vitamin C to prevent scurvy. Does he believe that the lesions which he got in the monkey are entirely due to deficiency of thiamine and not possibly modified by a deficiency of any other vitamin?

FOLLIS: I certainly feel that other factors which are missing in rice, namely poor quality or quantity of protein, lack of vitamins and of certain minerals, as well as absence of fat, may influence this picture. The presence of multiple deficiencies in rice is the reason for these experiments.

NADEL: Experimentally and in some autopsies in man, there have been described

TABLE 6. CARDIAC METABOLISM IN AN ALCOHOLIC WITH HIGH OUTPUT CARDIAC FAILURE

Substrate	Content Mg. % or MC %		Extraction A-V	Oxygen Extraction Equivalent %
	Bronchial Artery	Coronary Sinus		
Oxygen	8.3	2.1	6.2	
Pyruvate	1.5	1.5	0.0	0
Lactate	14.1	13.3	0.8	10
Glucose	86.4	84.0	2.4	29
Total				

cardiac lesions associated with excessive doses of desoxycorticosterone. In some cases animals have had cardiac lesions and ECG changes associated with potassium alterations. Do you believe that the long-term dietary deficiency that you exposed your animals to could of itself bring out in the animal a built-in 'stress-like' reaction associated with corticosteroid and electrolyte alterations that could give these heart changes—changes not specifically attributable to the vitamin deficiency we've been talking about today?

FOLLIS: This subject is an extremely important one. The first part of this year, I visited Dr. Selye's laboratory for several days. Both Dr. Selye and I have been particularly interested in myocardial necrosis on a nutritional basis for a number of years. When I showed him the cardiac lesions of one of the monkeys on a rice diet, I also told him that the animal had been tied down on a board that morning to take an x-ray and appeared perfectly well. When he saw the lesions, he suggested that they may have resulted from restraining the monkey on the board. Selye can do just this in rats. If he ties them down on a board for 24 hours, they show myocardial necrosis. However, we have seen this lesion since then in monkeys that have not been restrained on a board. In relation to the DCA experiments that you mentioned, the change is doubtless related to potassium deficiency which, as is well recognized, will produce exactly the same type of myocardial necrosis as does thiamine. Another sidelight on that may be of interest, however. When we were studying these two types of myocardial damage a number of years ago we thought that we could blow the rat's heart wide open if we put animals on a diet deficient in both potassium and

thiamine. But when we did this nothing happened (31). This brings up, of course, the tremendous importance of balance and imbalance on metabolism and growth which we're going to discuss a little more this afternoon.

HANDLER: Follis made a statement which surprised me, namely that the protein of rice is of poor quality. I thought quite the opposite was the case.

FOLLIS: It has been shown that supplementation of rice with small amounts of lysine improves the growth of rats (57).

HANDLER: Addition of lysine to almost any protein improves rat growth. The old data on the biological value of rice protein showed it to be about the best single protein available. And the data on humans treated with a rice diet for cardiovascular disease indicates that humans can apparently maintain nitrogen balance on a lower level of rice protein than on any other single known protein.

PATWARDHAN: I agree with the sentiments expressed by Handler. Of all the known cereal proteins which form the diet of the human being, rice, although it has less protein than many other foodstuffs, has protein of the highest quality for both the maintenance of nitrogen equilibrium in adults as well as for supporting growth in young growing animals. I think therefore that Follis might look into these facts and modify his statements accordingly.

OLSON: I have to come to the rescue of Follis; if you'll scrutinize the amino acid composition of rice protein fed at Kempner's level of 25 gm, methionine is definitely limiting. Maybe at higher levels this defect is overcome. But certainly the defense for rice might be made as a cereal protein, not as a total protein. Egg and casein are certainly superior to rice protein.

FOLLIS: Could Hundley discuss this? It's worthwhile to mention, Handler, that my statement was based on the situation when rice is fed alone, or even when it's fed with all the other vitamins and minerals and fat. I think the situation is a good deal different when you have the other deficiencies which are present in rice plus insufficient rice protein.

HANDLER: In the classical studies of the virtues of rice protein, separated rice protein was used just as we employ casein in the traditional rat diets. Under the circumstances, rice protein is of very high biological value. The problem here is not the amino acid composition as shown by analysis, but really its virtues in a diet. In the situation we are considering, you must take into account how many calories are being eaten per day, and how much protein will go with it. On a diet on which beriberi develops, I am not certain offhand what the total protein intake will be, but I know of no reason to think the quality of the protein is poor; rather does the total protein intake prove insufficient.

MACKENZIE: I'd like to ask Follis what evidence he has that the syndrome in these monkeys fed a rice diet is a thiamine deficiency.

FOLLIS: It has been cured with thiamine.

HUNDLEY: I would agree a little with both sides as to the nutritional quality of rice protein. In rats, one can improve the protein quality of rice by supplementing with small amounts of lysine and threonine (58, 59), just as one can improve the quality of almost any protein if one reduces the protein level sufficiently and uses the right amino acids. In adults there isn't much evidence as to whether these amino acids will improve protein quality of rice diets. We did some studies (60) in young adults and obtained very unpredictable responses. At times a favorable effect was observed on nitrogen balance with lysine or methionine. In other instances no effect, or adverse effects, were found. These experiments were done using diets supplying 26–30 gm of protein almost entirely from rice. In general, the results of this experiment indicate that the main shortage was simply utilizable nitrogen, essential or nonessential. The main defect of rice protein is the low level at which it occurs in rice. This becomes very important in trying to meet protein requirements from rice in young children.

I would also like to comment on questions raised about the specificity of the cardiac lesions in thiamine-deficient rats. I am confident that the lesions observed in our experiments can be attributed to thiamine deficiency specifically. The diets were supplemented with vitamins (other than thiamine), minerals, adequate fats, etc. We used pair-

fed controls that were subjected to the same stresses as the experimental rats, so that indirect stress effects through the adrenal seemed to be ruled out. We also analyzed cardiac muscle for sodium and potassium to rule out indirect effects of potassium. Some shifts in sodium and potassium were observed but these could not explain either the pathologic or electrocardiographic changes (61).

PLATT: We're getting very near the answers of these questions when we think of balance in the diet, and I would entirely agree with Handler on the protein point in relation to total calories, if he'd specify the origin of the remainder of the calories except the protein.

RAMALINGASWAMI: I should like to support what Hundley said regarding the relationship between B_1 deficiency and the nervous system changes. The work of North and Sinclair (45), which Follis referred to, is I think of particular significance here; in acute thiamine deficiency they did not find changes in the peripheral nervous system, but when they gave small doses of thiamine and prolonged the deficiency state they found characteristic changes in the nervous system.

NADEL: Follis, in the monkeys you had on this tightrope, when you gave them casein and pushed them off, is it the casein per se that did this, or would some other source of protein produce the same damage?

FOLLIS: Any material lacking in rice will. I'm going to discuss this more at length this afternoon, because I think it's an important consideration in relation to what Platt and Handler have mentioned.

OOMEN: Nobody wanted to say much in favor of the statement of Follis about the poor protein quality of rice, but I think I could because epidemics of beriberi are often seen when the rice supplies of the people are failing, then they use old, often molded rice, and I think in that case, the question of the protein presents quite another aspect of the question.

V. METABOLISM OF THIAMINE

HANDLER: As a biochemist, who is proud of the accomplishments of modern biochemistry, it is a humbling experience to talk about thiamine metabolism before this group. It would be pleasant if we had information which would permit an explanation in molecular terms of the problems which were raised this morning. Unfortunately, we cannot.

With your permission I shall not attempt a scholarly review of thiamine metabolism, but, rather, confine my discussion to what is really known of the answers to a group of simple questions. First, what do we know about the biosynthesis of thiamine? Second, what is known about the degradative fate of thiamine in mammals? Third, in what reactions does thiamine participate catalytically and how does it effect such catalyses? Finally, can we relate any of these observations to the manifestations of thiamine deficiency? With the exception of question three, for none of these have we reasonable answers.

Thiamine Biosynthesis. The literature in this area is quite sparse. There are enzymes in various plant materials and in rabbit liver and kidney which are capable of effecting a union of the thiazole and pyrimidine moieties of this vitamin, but the biogenesis of the thiazole and pyrimidine moieties is completely obscure. This is rather remarkable at a time when tracer materials are easily available; yet there have been no recent recorded attempts to study this problem.

The Fate of Thiamine. There is an abundant literature describing balance studies and the quantitative excretion of various materials which appear in the urine after thiamine administration. Yet we know nothing of the real fate of thiamine. It is apparent that thiamine itself appears in the urine of individuals on ordinary rations and that the amount increases after thiamine administration. But most of the ingested thiamine does not reappear in the urine as such, and we have no adequate description of what happens to the rest. We are unaware of any mammalian enzymes capable of degrading thiamine itself. However, when thiamine, labeled in the thiazole ring with C^{14} or S^{35}, was administered to rats, and their urine examined by paper chromatog-

raphy, a total of 16 separate radioactive spots were detected on the paper. The identity of few of these materials was established. There was clearly some thiamine, as expected, as well as thiamine disulphide in which the thiazole ring is opened, and the sulfhydryl groups of two thiamines which are coupled by a disulfide bridge. There were also a number of compounds which, presumably, were mixed disulfides in which the thiamine sulfur is joined to other sulfhydryl compounds e.g., coenzyme A, cysteine or glutathione. They are probably artifacts rather than enzymatic products. Whether they have any physiological meaning is unclear. Thus, the enzymes responsible for degrading thiamine have not been identified, if such there be.

As you know, thiaminase is distributed in rather limited fashion. It is found in certain fresh- and salt-water fish and it has also been found in bracken ferns and in molluscs. No equivalent enzyme is known to be present in mammalian tissue, however. Thiaminase is a rather remarkable enzyme, of interest to the biochemist for its own sake. It effects an exchange; aniline, for example, may exchange for the thiazole portion of the molecule, giving rise to a compound which is then catalytically inert. The role of this enzyme in the relatively rare places in which it has been found is not known, but it is conceivable that ordinarily it serves not to destroy thiamine but for thiamine synthesis, and may normally put together the thiazole and pyrimidine portions of the molecule. The same enzyme has been found in a few microorganisms which have been isolated from the stool of humans. This work has been done in Japan (62). Two bacilli and one clostridium have been so recognized, all isolated from persons said to have beriberi. According to one report such organisms can be isolated from the stools of 3% of a normal population and those of 15% of the beriberi population which were examined. If these bacteria were present in high numbers in the intestinal tract, they might result in the destruction of dietary thiamine. It seems unlikely that these organisms play a significant role in the pathogenesis of beriberi. Indeed, they would only be expected to flourish in the intestines of persons eating abundant thiamine. Thus the fate of thiamine in man remains an open question.

The only enzyme known to affect thiamine in animal tissues is that which converts thiamine into cocarboxylase. This enzyme, which promotes a reaction between thiamine and adenosinetriphosphate, moving the pyrophosphate moiety of the ATP onto the thymine with formation of thiamine pyrophosphate and adenylic acid, has been isolated and partially purified from intestinal mucosa. There have been statements that thiamine monophosphate can be converted to the pyrophosphate, but only the enzyme mentioned above is known with certainty. Incidentally, it is one of very few known instances in which the terminal pyrophosphate of ATP is transferred to an acceptor molecule.

Thiamine pyrophosphate can be degraded by a number of pyrophosphatases, and thiamine monophosphate, should it ever exist, is readily hydrolyzed by various nonspecific phosphatases. This is really all we have to say about the synthesis and metabolic fate of thiamine; little else is known with any certainty.

The Role of Thiamine. And so we move to the area which biochemists know best, the role of thiamine in intermediary metabolism. In mammalian metabolism, thiamine pyrophosphate is known to participate in three reactions. Thiamine pyrophosphate is necessary for the opening step of the reactions which feed into the citric acid cycle. Here, pyruvic acid is decarboxylated and 'active acetaldehyde' exists transiently on the thiamine before it is transferred to the oxidized form of lipoic acid. The lipoic acid is thereby reduced and acetyl lipoic acid comes into being. This is the fate of pyruvic acid which is dominant in the oxidative metabolism of mammals, and this is the role of thiamine which was first recognized in mammalian systems. The simple decarboxylation of pyruvic acid to acetaldehyde, the first thiamine-dependent reaction ever observed, never occurs in mammalian metabolism. The formation of *free* acetaldehyde by decarboxylation appears to be restricted to microorganisms.

There is a second reaction, entirely analogous to that in which pyruvic acid engages. This is the oxidative decarboxylation of

d-ketoglutaric acid, a later step in the citric acid cycle series. The role of cocarboxylase in this reaction resembles exactly its participation in pyruvic acid decarboxylation. The *d*-ketoglutaric acid is decarboxylated and 'active' succinyl semi-aldehyde exists in transient fashion on the thiamine and then is transferred to lipoic acid, giving rise to succinyl lipoic acid from which the succinyl group is transferred to coenzyme A.

The mechanism of these two reactions is presumed to be identical but has not been definitely established. I have no brief for one mechanism as compared with the other.

More recently a third reaction has been described, which may not be so well known to any of you; this is the transketolase reaction. As you may know, there is a system of reactions which has been called the 'direct oxidative pathway' of glucose metabolism. That such a pathway exists was indicated by the work of Warburg, Lipmann and Dickens over a period of about 25 years, but that there is a working pathway was never demonstrated. The initial reaction in this sequence is the following: glucose 6-phosphate + TPN → 6-phosphogluconic acid + TPNH. It was because of this reaction in red cells that TPN was found by Warburg. And since this reaction occurs in liver, red cells, yeast, brain and adrenal cortex, it had to be significant. More recently, the next step in this series was found: 6-phosphogluconic acid + TPN → ribulose-5-phosphate + CO_2 + TPNH. Ribulose is a 2-ketopentose. These are the opening steps of the direct oxidative pathway.

Our understanding of the succeeding steps in this sequence is due largely to the work of Horecker at the National Institutes of Health and Racker of the Public Health Research Institute of the City of New York. The operation of the sequence may be summarized as follows: assuming the glucose to have been phosphorylated by ATP in the presence of hexokinase as in the usual glycolytic sequence, and starting with 6 moles of glucose, the combined reactions thus far can be represented as:

6 glucose + 6 ATP + 12 TPN
→ 6 ribulose-5-phosphate + 6 CO_2
+ 12 TPNH + 6 ADP

Thus the equivalent of one glucose molecule has been completely oxidized and its reducing potential now resides in the 12 TPNH. The object of the remainder of the process is to perform the overall conversion

6 ribulose-5-phosphate
→ 5 glucose-6-phosphate

Among the enzymes necessary is *transketolase* which catalyzes the general reactions: xylulose 5-phosphate (a ketopentose), ribose 5-phosphate, 3-phosphoglyceraldehyde and sedoheptulose 7-phosphate (a ketoheptose). Thiamine pyrophosphate is coenzyme for this reaction and there exists an intermediate 'active glycolaldehyde' analogous to the previously mentioned 'active acetaldehyde'. A balanced scheme for the entire sequence has been described as the 'direct oxidative pathway', which occurs not in the mitochondria but in the cytoplasm of cells, including those of liver, brain, adrenal cortex and kidney. Skeletal muscle is the one place where this pathway is not operative. This pathway is the only known mechanism for synthesis of ribose for nucleotide formation and, at least equally important, results in formation of TPNH which is necessary for the operation of a number of systems, such as hydroxylation of steroids. Moreover, TPNH is necessary for the reduction of intermediates from carbohydrate metabolism to form fatty acids.

These then, are the three reactions in which thiamine pyrophosphate participates. In addition, there is one other reaction which is of some interest. As Mackenzie indicated, our concept of alcohol metabolism is that the first step is the oxidation of alcohol to acetaldehyde by alcohol dehydrogenase which is widely distributed. This acetaldehyde may suffer two alternate fates insofar as we are aware. In the first, the acetaldehyde is oxidized to acetic acid by either xanthine oxidase or the aldehyde oxidase of liver. Both are flavoproteins and no useful energy is derived therefrom. In the other path, acetaldehyde can serve as an acceptor for active acetaldehyde when the latter comes from pyruvate. Instead of transferring the acetaldehyde to lipoic acid, as one ordinarily does, acetaldehyde can be used as an acceptor by the active acetaldehyde which is

formed from the oxidative decarboxylation of pyruvate. This gives rise to acetyl methyl carbinol (acetoin). When alcohol is administered, acetoin is always observed in blood and in urine. This is perhaps an accident, rather than a normal metabolic pathway. To the extent to which acetaldehyde is converted to acetic acid effectively, it enters the pathways of fat metabolism. To the extent to which this other reaction occurs it seems to be a dead end and acetoin may not suffer any useful subsequent metabolism. On the other hand, when a load of alcohol is imposed upon the organism, it may be necessary to use this device lest acetaldehyde accumulate in quantity.

Well now, can we provide any basis for an understanding of the manifestations of thiamine deficiency in the light of this information? You know the answer is "No". From current knowledge of intermediary metabolism we could not possibly predict that thiamine deficiency would result in beriberi, nicotinic acid deficiency in pellagra and riboflavin deficiency in the syndrome of ariboflavinosis. Moreover, current literature reveals very few experimental attempts to elucidate these relationships.

Let us, therefore, consider the possible mechanisms by which thiamine deficiency might result in beriberi. Do manifestations of thiamine deficiency represent a failure of nervous tissue to obtain sufficient energy? The object of burning glucose is not to make carbon dioxide. Presumably, it is to provide ATP as an energy source. Is there a failure to provide sufficient energy in a cell which lacks sufficient thiamine pyrophosphate? Please note the tacit assumption that it is *lack* of thiamine pyrophosphate which is here involved. If simple failure to obtain energy were involved, then why are not the manifestations of thiamine deficiency, of nicotinic acid deficiency and of riboflavin deficiency all identical? All of them would be reflected in a failure to make ATP. The second possibility is that, among the reactions catalyzed by thiamine pyrophosphate, one or more yield products which are uniquely necessary for the metabolism of heart or of nerve. Then, what we are witnessing is failure to deliver a nutrient, in this sense, to the cell. Again there is no demonstration that this is the case.

The third possibility is that, for lack of thiamine, a reaction does not proceed at a sufficient rate and, in consequence, something accumulates which is toxic. Much attention has been paid over the years to pyruvic acid. Strangely enough, I know no description of what happens if one gives large quantities of pyruvate to the depleted animal who is not yet moribund and shows no signs of polyneuritis. What happens if you overload him with this material which, supposedly, he cannot metabolize? There is another compound which has caught my attention. In the literature there are repeated statements to the effect that the deficient animal accumulates modest amounts of methyl glyoxal. This statement has appeared rather frequently in the literature and I presume that it is correct. We have no information concerning the metabolic origin of methyl glyoxal, although we do know of a pair of enzymes which together are called glyoxalase and catalyze the reaction: methyl glyoxal → lactic acid. However, the coenzyme for this system is not thiamine pyrophosphate but glutathione. It is not entirely certain that there is no cocarboxylase on the enzyme in addition to the necessity for glutathione. But the possibility that a local rise in methyl glyoxal concentration may be of significance in the pathogenesis of beriberi seems attractive and warrants further investigation.

With respect to the problems posed this morning regarding Wernicke's syndrome and the Korsakoff psychosis and the relationships of alcohol to these, there is clear evidence that the acute tolerance of vitamin B_1-deficient animals to doses of alcohol is considerably below that of normal controls. Moreover, acetaldehyde is considerably more toxic to the thiamine-depleted animal than is alcohol itself. This fits the concept stated above that it is the metabolism of acetaldehyde for which thiamine is required, rather than that of alcohol itself. There's one more problem which warrants our attention. This is the thiamine-sparing action of dietary fats, first described by Westenbrink. It has been studied frequently since, and the reason for calling it to your attention is that it ought to afford a useful clue to the factors which determine the loss of thiamine from the animal. There are several reasons

why a diet high in fat would spare the requirement for thiamine. The first of these is obvious. Of the three reactions we have described in which cocarboxylase participates, only one is required for the further oxidation of fat and this is the oxidation of *l*-ketoglutarate. If fatty acids rather than glucose are the prime source of calories, then the animal might get by with just enough thiamine pyrophosphate for this one enzyme. No one has yet measured the disappearance of transketolase itself in the thiamine-depleted animal, but it is known that the pyruvic decarboxylase activity declines well before the *d*-ketoglutarate decarboxylase activity declines. This is in keeping with the fact that the thiamine-deficient animal can handle fat more successfully than carbohydrate. That the direct oxidative pathway is markedly affected by thiamine deficiency has been demonstrated only in red cells. In the red cells of B_1-deficient birds, presumably the transketolase reaction slows down and the ability of these red cells to produce carbon dioxide from glucose may be as little as one-seventh of normal. Under these circumstances pentoses increase in these cells by a factor of about three. The accumulation of methyl glyoxal may also relate to this failure. The next possibility is that there may be a toxic product derived from carbohydrate metabolism and not from fat metabolism. Here again we may reconsider methyl glyoxal which, presumably, does not arise out of fat metabolism. The third possibility is that fat somehow 'protects' thiamine from destruction or loss from the body. The pertinent studies in this regard, however, indicate that at marginal levels of thiamine consumption more thiamine appears in urine if the diet is rich in fat than when carbohydrate is the main source of calories. This in turn almost suggests that 'unused' coenzyme is discarded, a rather surprising notion. I have no mechanism to offer which might indicate how this might operate.

Finally, there is the information which has come to us from the use of thiamine antimetabolites. As far as I can see, oxythiamine and pyrithiamine are potentially useful experimental tools but have shed no light on the problems which we have posed here. These antimetabolites accelerate the course of thiamine deficiency in animals. Some confusion was created by indications that the pyrithiamine-treated animal develops a polyneuritis without showing an impaired carbohydrate oxidation. This led to the suggestion of a special role of thiamine in the metabolism of nerve cells quite independent of the three reactions we have considered. However, others have challenged this notion, as they have found that polyneuritis is observed in the pyrithiamine-treated bird only when there has been a substantial decrease in the cocarboxylase of whole brain.

Thus, no satisfactory account can be given of the metabolic basis for the manifestations of thiamine deficiency.

HUNDLEY: If I understand Handler correctly, he stated that alcohol was toxic to the B_1-depleted animal. I wonder how he reconciles this with the fact that seems to have been clearly demonstrated that if the portion of the carbohydrate calories is replaced by alcohol, the onset of polyneuritis is delayed by a considerable margin.

HANDLER: I can rationalize that, if you wish. You will recall that what we said is that alcohol going to acetaldehyde and then on to pyruvic acid metabolically is equivalent to giving fatty acids. If the alcohol intake is low, acetaldehyde never accumulates in sufficient quantity to be acutely toxic. Then you would really have substituted fat for carbohydrate in the diet and it really should prolong the animal's life. The dose of alcohol isn't of such magnitude as to make a substantial accumulation of acetaldehyde. This is pure rationalization. My previous statements related to studies of acute toxicity.

VI. Special Problems Concerning Beriberi

A. *The Neurological Aspects of Thiamine Deficiency*

DENNY-BROWN: It is remarkable how severe and prolonged simple starvation can be in man or animal without damage to the nervous system. In some instances dark staining of myelin by the Marchi method has been described, but there are no lesions that

the neuropathologist would accept. The muscles may become extraordinarily thin with surprising retention of power of contraction. There is surely no doubt of the relationship between beriberi and polished rice, or correction by rice polishings or by pure thiamine. The difficult questions are the explanation of the delay in recovery after prolonged paralysis, the variability of syndromes in similar circumstances, and, more cogent in this climate, the relationship of beriberi to conditions such as alcoholic and other polyneuropathy and encephalopathy.

From the neurological point of view, the most important feature of beriberi as I saw it in prisoners of war (63) is symmetrical foot drop and wrist drop, associated with great tenderness of muscles and a mild disturbance of general sensation over characteristic areas over the outer aspect of the legs, the thighs, and in patches over the abdomen, chest and forearms, as pictured in the diagram of Miura (64). Sometimes there is sensory loss in the lips. Absence of ankle jerks, pains and discomfort in the feet, muscle cramps and paresthesias, though common in beriberi, cannot be accepted as criteria when these above are present, for each of these has so many possible causes. Ataxia with loss of position and vibration sense should not be early signs of beriberi, and should lead to suspicion of pellagra. Cardiac changes of some degree were universally present in cases of beriberi observed by us, but sometimes persistent tachycardia was the only evidence of these. When beriberi was particularly acute, aphonia from laryngical paralysis, and, sometimes, deafness would occur.

That alcoholic polyneuropathy could be comparable to beriberi was suggested by Shattuck (65), and proof of improvement in such cases by the administration of thiamine during continuation of alcohol and previous diet was obtained by Strauss (66, 67) in 1935. For a time it was assumed that all alcoholic polyneuropathy was due to thiamine deficiency. The demonstration by Peters and his associates (68) that arsenic interfered with the pyruvic acid stage of the carbohydrate cycle suggested that this was a common metabolic link for neuropathic disturbances. The acute appearance of foot drop certainly occurs in alcoholism but it is not the only form of alcoholic polyneuropathy. More common is a chronic sensory neuropathy, with more disturbance of pain sensation than occurs in beriberi and prominent burning paresthesias in the feet. This is also characteristic of chronic diabetic neuropathy, pellagra, and in other coditions. The Japanese (69) had claimed that ataxia and amblyopia occurred as part of beriberi; our own experience in World War II made it clear that these disorders could occur with other symptoms of pellagra when beriberi was not prevalent. The tropical experience of Scott in Jamaica, Pallister in Singapore, Stannus in Africa and Metivier in Trinidad summarized in our earlier paper (63) had in fact shown that various combinations of the ataxia-amblyopia-burning feet syndrome could be independent of beriberi. Ataxia and loss of vibration and of position sense appear in a number of different types of neuropathy, among which that related to carcinoma of lung is of special interest, but they have no relation to beriberi. For this reason I emphasize the foot drop and wrist drop as features of the beriberi syndrome, for these clearly distinguish it from other types of neuropathy.

In these latitudes the beriberi syndrome is usually associated with moderately severe dietary restriction associated with alcoholism. With the most severe restriction, as in a person who has literally lived on alcohol for 2 or 3 weeks, the outcome is Wernicke's encephalopathy. Various mixtures of polyneuropathy and Wernicke's syndrome are well described by Victor and Adams (70). Most commonly the neuropathy develops while the patient is under ineffective treatment for encephalopathy. These combinations can occur with any type of severe recurrent vomiting such as the pernicious vomiting of pregnancy, or of pyloric obstruction (71). One of the original cases of Wernicke was a patient whose vomiting was due to pyloric obstruction. Well known also is the precipitation of encephalopathy in these circumstances by overloading carbohydrate metabolism by the administration of large amounts of glucose without the proportional thiamine coverage. Acute high output cardiac failure can be precipitated in the same way.

I think we need to define carefully what we mean by 'fulminating'. An intercurrent infection, vomiting or some other stress may produce acute symptoms on a background of a chronic deficiency. Encephalopathy requires a more complete deprivation than is usually accomplished by eating polished rice. I think this is the answer to Platt's question this morning. When the critical stage of severe thiamine depletion is imminent, sudden death from cardiac failure is well known in Wernicke's syndrome, and is in fact the usual cause of death. The more common occurrence of cardiac symptoms than encephalopathic in Oriental beriberi may therefore be related not only to severity of depletion but also to factors favoring congestion of the venous circulation in warm climates. The seasonal incidence, and Platt's graphic description of the onset of Shōshin with the restlessness and insomnia of the first humid nights of the hot weather, particularly favors such secondary determinants of cardiac rather than neurological symptomatology.

I would summarize these aspects of thiamine deficiency by listing the symptoms in order of acuteness of deprivation. The most acute symptoms I would list as mental confusion leading to coma. With these, depending on how rapid is the onset, appear the bilateral 6th nerve weakness. The combination is then Wernicke's syndrome. In lesser degree the 6th nerve weakness becomes simply nystagmus on looking to either side. In still lesser degree aphonia with general mental confusion, may be the first or perhaps only symptom. As we go further down the scale of intensity, mental confusion becomes associated with varying degree of foot drop-wrist drop syndrome, appearing after a more prolonged deficiency state. Finally, the most chronic you would recognize would be foot drop alone, evolving slowly on a background of very chronic deficiency. Of these symptoms the 6th nerve weakness is the most easily removed by administration of thiamine (72), mental confusion and ataxia being quite difficult to reverse. Wrist drop and foot drop can be reversed with ease or difficulty according to their duration. The Korsakoff syndrome commonly appears in relation to the subsidence of mental confusion of Wernicke's syndrome, and we believe that it represents a milder degree of persistent confusional state of that syndrome (73).

Now I would like to turn to pathology. The remarkable feature of experimental thiamine deficiency in the pigeon has always been the opisthotonos, its reversibility, and the absence of histological lesions in the brain stem. I think the key to the proper understanding of these disorders is the work of Swank (74). Swank showed that the symptomatology depended on the degree of deficiency; that by adding minimal amounts of thiamine to a totally deficient diet, the symptoms changed. The most severe deprivation led to the sudden onset of gross disturbance of righting reflexes and other function of brain stem leading to rapid death. With less reverse deprivation, there was involvement of cranial nerves with lesions appearing in the tegmentum of the medulla. With the less complete deficiency still leg weakness appeared. If more thiamine still was added to the diet, leg weakness alone could be regularly produced. He also showed that in the most acute type, there was no demonstrable histology at all in the nervous system. With less acute there was damage to basal nucleii and in chronic forms degeneration of peripheral nerves was produced. The distal parts of the longest nerve fibers were affected first. Each of these types could be reversed by adding thiamine to the same diet. Swank also noted chromatolysis of nerve cells and focal necrosis of the myocardium in moderately acute states, whereas no change in cardiac muscle is found in the most acute stages.

In Wernicke's encephalopathy the histological changes are chiefly in the nature of capillary damage in the mammillary bodies, the related walls of the third ventricle, the aqueduct, and in the tegmentem of the medulla. When the condition is severe and acute there are pin-point hemorrhages in all these situations. Sometimes retinal hemorrhages of the same kind are seen. The most prominent histologic change is capillary damage, with endothelial proliferation and microscopic hemorrhage. In more chronic states brown staining, loss of ground substance, endothelial proliferation and ischemic changes in related nerve cells are

seen. In the medulla the region of the nuclei of the vagus is more often affected than the nuclei of the 6th nerves. The 6th nerve paralysis has no histological correlation, and it is clear that the loss of awareness and ataxia represent a disturbance of function of brain and cerebellum that is very much more widespread than the histological changes indicate. The cerebral cortex may appear edematous but show no other change. If arsenical encephalopathy can be accepted as the maximal degree of disturbance of the pyruvic acid stage of carbohydrate metabolism, the widespread purpura of the brain seen in that condition would represent more adequately the distribution of disturbance in function that in Wernicke's encephalopathy occurs as a 'biochemical lesion', progressing to histologic lesion only in the mammillary and related areas.

It is only in the more chronic states, or after repeated episodes of Wernicke's syndrome, that nerve cell changes become general. They then take the form of partial chromatolysis with displacement of the nucleus to one side of the cell that was described as 'central neuritis' by Adolf Meyer (75). This is a nonspecific type of cell change and peculiar to states such as Korsakoff's psychosis (76) only in its generalization in the nervous system. It differs from the similar change in pellagra only in that with Korsakoff's psychosis the cell is usually darker and more glassy in appearance.

At this point I should comment that 'convulsive seizures' described in thiamine deficiency in the rat do not necessarily imply cortical involvement. In such animals acute disturbance in brain stem function can result in paroxysms of running and turning pseudo-convulsive movements.

In the peripheral nerves we find changes of considerable interest. Severe beriberi is associated with Wallerian degeneration beginning in the most peripheral part of the longest nerve fibers. These have been well described in the early studies of Pekelharing and Winkler (77) and in the experimental animal by Swank (74) and others. Pekelharing and Winkler noted a peculiar segmental thinning of myelin more proximal to the areas of nerves that show Wallerian degeneration. This is the early and reversible stage of the lesion. Segmental damage to myelin has been known for a considerable time amongst those who have studied peripheral nerve lesions as the 'segmental neuritis' of Gombault, and has been described in a variety of peripheral neuritides. The myelin is thinned or lost for part or the whole of a segment without destruction of the axis cylinder. This change is seen commonly by us in the beriberi type of alcoholic polyneuropathy in nerves such as the anterior tibial. The defective segment of myelin is from .1 to 1 mm in length. Commonly the myelin is beaded. You will note the complete absence of the oval encysted globules of free fat that are characteristic of true Wallerian degeneration. Some histiocytes in the area contain free fat. The axis cylinders stain poorly and are irregular in outline when they traverse the affected area. In the smallest peripheral nerves in chronic degrees of the syndrome the beading and distortion of axis cylinders and paucity of myelin may be very striking. Small gaps in myelin are commonly found in other types of multiple neuritis, such as diphtheritic and diabetic neuropathy. It is also seen in the dorsal roots in combined system disease (B_{12} deficiency). What is so characteristic of beriberi is the presence of such demyelination in long segments in the most peripherial parts of the peripheral nerves. In a more patchy fashion it will occur in porphyria.

This segmental lesion of myelin can be produced experimentally by ischemia (78). There is then a gap in myelin in every fiber as it passes through the region rendered ischemic, with failure of motor conduction but not sensory. Such a nerve block produced experimentally by ischemic anoxia lasts about 19 to 21 days. There then appears a thin layer of lipid and conduction is renewed, but even after 6 weeks there is still only a thin myelin coating of the previously blocked segment.

In summary, then, the neuropathology of thiamine-deficient states is a spectrum of changes running from maximal disturbance of function with minimal histologic change, to chronic states with minimal biochemical change and well defined peripheral nerve lesions. Beriberi occupies one band in that

spectrum, Wernicke's encephalopathy another. In this connection it is of interest to contrast B_1 deficiency with B_6 and B_{12} deficiency. The essential feature of demyelination with preservation of the axis cylinder is fundamentally similar in all these. It is the location of the lesion that differs. The B_1 deficiency is the distal part of the peripheral neurone, motor more severely affected than sensory. In pyridoxine and pantothenic acid deficiencies it is the dorsal nerve roots that suffer (79), just as in B_{12} deficiency it is the dorsal and lateral columns of the spinal cord and to a lesser degree the dorsal nerve roots (80). In each of these deficiencies the most severe type of deprivation results in cerebral neuronal symptoms, without specific neuropathology. Mental confusion in thiamine deficiency, mental confusion and epilepsy in B_6 deficiency, and mental confusion and paranoia in B_{12} deficiency (combined system disease). In each case the lesser degrees of experimental deprivation result in peripheral nerve degeneration and the most severe degree of restriction (as produced by vitamin antagonists) in cerebral damage. The cerebral lesions of B_{12} deficiency have now been produced in animals by a B_{12} antagonist (81). Combined system disease is just one part of the spectrum of B_{12} deficiency, just as beriberi is only one part of the spectrum of thiamine deficiency. Thiamine alone causes capillary damage when acute, and I believe this sets it apart from these other types of disorder.

It was hoped that study of blood pyruvic levels in man might lead to some measurable objective index of thiamine deficiency. Raised pyruvic blood levels have been found in experimental beriberi in the pigeon by Thompson and Johnson (82) and in the natural tropical beriberi by Platt and his colleagues (4); estimates in clinical neuropathy in temperate climates have given equivocal results. Bueding *et al.* (83) found elevated fasting values in acute alcoholic neuropathy and encephalopathy but no significant change in chronic polyneuropathy. This, in general, has been the experience of others (84). If I might highlight the neurological problems of thiamine deficiency as they appear to me, I would first point to our present inability to account for the change in place of lesion in the nervous system, ranging from damage to capillary endothelium of brain in acute state of deprivation, to damage of nerve cells, then to selective myelin damage in peripheral nerve in the chronic states. Our own experiments of brain infarction have shown that the capillary endothelium of brain is as easily damaged by anoxia as is the parenchyma, and in some situations more so. If the circulation continues, capillary hemorrhages then occur. The occurrence of widespread purpuric hemorrhage with some types of acute thiamine deficiency compared with death without lesions in the most acute states may therefore be only a matter of duration of continuing circulation after damage to capillary endothelium. If death occurs sufficiently rapidly, as in the completely thiamine-deficient pigeon, no histological change other than edema may then be seen.

B. *Infantile Beriberi*

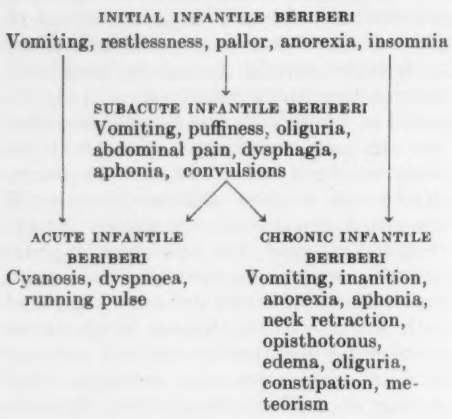
BURGESS: Most of the pre-war records of beriberi and such post-war records as there are, refer almost exclusively to the disease in the adult. I think that it is true to say that there has never been an adequate assessment of the importance of beriberi as a cause of mortality in the infant and that this aspect of the total problem urgently needs clarification.

The adult form of beriberi has been recognized for many centuries, but it was only towards the end of the last century that Hirota (85) described a clinical entity in the infant which he recognized as beriberi. After this, infantile beriberi received quite a lot of attention in Japan, though it does not appear to have been of major significance as a cause of death. In 1925, when the total incidence was high, only 3.5 infant deaths per 1000 births were attributed to beriberi (86).

In the Philippines, however, the infantile form of the disease was thought to be the main cause of the very high infantile mortality, and much of the early work was done there. In 1904 Guerrero (87), on the basis of clinical observations, concluded that the disease known as 'taon', which was responsible for such a large number of infant deaths, was in fact beriberi.

Subsequent investigations carried out by

TABLE 7


INITIAL INFANTILE BERIBERI
Vomiting, restlessness, pallor, anorexia, insomnia
SUBACUTE INFANTILE BERIBERI
Vomiting, puffiness, oliguria, abdominal pain, dysphagia, aphonia, convulsions
ACUTE INFANTILE BERIBERI
Cyanosis, dyspnoea, running pulse
CHRONIC INFANTILE BERIBERI
Vomiting, inanition, anorexia, aphonia, neck retraction, opisthotonus, edema, oliguria, constipation, meteorism

McLaughlin and Andrews in 1910 (88) verified this conclusion. They performed some 219 autopsies in infants under 1 year and found that 56% had died of beriberi. Prior to death, many of these had not been diagnosed as suffering from beriberi. Many cases in which the cause of death was given as broncho-pneumonia proved in fact to have died from beriberi. They concluded that the very high mortality rate in the early months of life in the breast-fed infant was almost entirely due to this disease. They pointed out that at that time in Europe and North America, it was the artifically-fed infant who died, while the breast-fed baby in the first few months of life was generally healthy and well nourished and that nowhere else had nearly such a high mortality been recorded in breast-fed infants. The infantile mortality rate in that year in Manila was 448 per mille. In 1912, Andrews (89) recorded further observations. He laid stress on the age at death which is an important characteristic of the disease. He found that nine-tenths of the deaths occurred between the ages of 1 and 3 months, although he saw cases aged 4, 5, 6 and even 7 months old. Once or twice he saw the disease in infants at 10 months of age. His paper contains an excellent clinical description and he draws attention to the fact that the onset of the disease is often not perceived by the mother because the early signs are mild in nature. When the frank condition develops, the course is rapid and frequently ends fatally. Andrews also records the occurrence of sudden death. He showed that the maternal milk was responsible for the death of the infants, for he was able to produce beriberi in puppies by feeding them on the milk of women whose infants had died of beriberi.

It was about this time that Vedder and Chamberlain (90) developed an extract of rice polishings known as 'tikitiki' extract, which proved effective in the treatment of the mild case and was later used in prevention.

The problem of infantile beriberi in the Philippines was followed closely for many years by Albert, who published his first paper—an account of a typical case of beriberi with autopsy findings—in 1908 (91), and his last on infantile beriberi in 1947, the year he died (92). In 1931 he reported that the introduction of the extensive use of 'tikitiki' had reduced the infantile beriberi mortality from 93.66 per 1000 births in 1915 to 19.12 per 1000 births in 1930. In 1933, 1934 and 1935 there was an increased incidence. In the latter part of the war period, and immediately after the war, no cases were admitted to hospital. However, in 1947 cases again appeared. In his survey of beriberi in the Bataan, Salcedo (93) found few cases of infantile beriberi in his clinical survey of the population, in spite of the fact that the majority of the total deaths from the disease occurred in infants.

There are reports from a number of other countries indicating that infantile beriberi was an important cause of morbidity and mortality. Bray (94) described the disease in a small population under medical supervision in Nauru Island in the Pacific. The age incidence was largely confined to the 9th, 10th and 11th week of life and was responsible for a high proportion of the total deaths. It was usually an acute condition which was rapidly fatal. The mothers generally showed little or no sign of the disease.

In Malaya, frequent reference is made in pre-war reports to the contribution made to the high infantile mortality by this disease, but no detailed investigations have been reported. Haridas (96) records the admissions of over 300 cases to the children's wards in the General Hospital, Singapore.

These constituted nearly 11% of all admissions from all causes to these wards. Mention is also made by other pediatricians of the frequency with which the disease was seen in this city (97).

Aykroyd and Krishnan (98) drew attention to the high infantile mortality rates in certain areas in what was then called Madras Presidency. The characteristic of this infantile mortality was the high proportion of children dying between 2 and 5 months with a peak being reached at 4 months. The authors considered that this evidence was suggestive of a high death rate from beriberi but did not constitute proof of it.

Krishnan, Ramachandran and Sadhu (99) treated over 1000 cases of infantile beriberi in the same area, but did not attempt to find out the proportion of infants that developed beriberi or the quantitative effect of the disease on the infantile mortality rate.

In Hong Kong immediately before the war infantile beriberi was said to be very common and was believed to be largely responsible for the excessively high infantile mortality rate of over 300 per mille. Fehily (100) states that 18% of the infants attending infant welfare centers showed signs of infantile beriberi and that the mortality rate in untreated cases was over 90%.

In the Netherlands Indies and Indochina, before the war, existence of a health problem produced by infantile beriberi was recognized. In a recent report from Vietnam, Tran Van Tu (101) records some 273 deaths, of which 128 occurred in the 2nd and 3rd months of life. He believes the disease to be responsible for many infantile deaths in that country.

Sharma (102) describes the many cases of infantile beriberi which he saw during his 40 years as a pediatrician in Burma, and the practices and dietary habits which were their unvarying accompaniment. Reports from a WHO consultant support the view that beriberi is an important cause of infant deaths in that country.

I do not suggest that this is a comprehensive review of what has been published on the incidence of infantile beriberi in a thiamine-deficient population, but I have not been able to trace any other accounts which define any more accurately the effect of the disease on the total infantile morbidity and mortality.

Some of the publications to which I have referred suggest that a very high infant death rate results, and it may be that this can occur under some circumstances and not under others. This is clearly a matter on which we must have exact information. Quite apart from the desirability of understanding a problem thoroughly before suggesting measures for its solution, the introduction and maintenance of preventive measures may present considerable difficulty, and it is essential that the governments of the countries concerned should have a clear idea of the national significance of the disease. Preventive measures may involve interference with trade, the introduction of legislation and/or the expenditure of considerable sums of money, and governments must be convinced that such measures are necessary and valuable.

It may well be asked why we are unable to define our problem more accurately at the present time. It must be remembered that in the pre-war period the registration of births and deaths was far from complete in most of the countries affected and there was therefore no reliable information as to the total number of infants dying. Also, at that time, the attention of health authorities was concentrated on communicable disease which was an ever-present menace to the health of the total population. In addition to this, the people among whom the disease most often occurred were generally unsophisticated, little exposed to modern developments, and naturally inclined to consult their own traditional healers rather than the trained practitioner. Lastly, there appears little doubt that the disease was often not diagnosed.

At the present time some of these circumstances remain. Reliable figures for infant mortality rates are not always available and some hesitancy in consulting western trained medical men still exists, especially in the rural areas.

Moreover, it is essentially the rural populations that are now being exposed to the risk of an increased incidence of both adult and infantile beriberi. There, following the spread of small rice-milling machines

throughout the countryside, the change over from an under milled to a highly milled rice is taking place—and this is happening in populations which derive a very high proportion of their calories from rice. The medical services to these communities are limited and, as I have already said, may not be easily accepted. In any case transport is difficult owing to the inadequacy of the road system. Rural people in rice-growing countries usually live in small villages approachable only over paths on the tops of the bunds in the rice fields, which in the wet weather may be flooded and are always difficult to negotiate. This last point is one of considerable importance, because beriberi is a seasonal disease in many countries—a higher incidence occurring during the rainy season. Under these circumstances there is likely to be difficulty and delay in getting children to the hospital or clinic.

Now infantile beriberi is often an acute disease. Andrews (89), Bray (99), Fehily (100) and Sato (103), among others, have stressed the general acute nature of the disease and the frequent occurrence of sudden deaths. The last writer regards sudden death in the breast-fed infant as being pathognomonic of infantile beriberi.

Haridas (95), in a review of 108 cases, found signs of cardiac disease in 94 of them. Even with treatment he had a high death rate. In circumstances of comparable severity in remote villages with poor communications, it is unlikely that any of the affected children would be seen and the disease diagnosed. It is therefore perhaps not so difficult to understand why, so far, we have not been able to make a reasonably accurate assessment of the situation.

However, it is not nearly so difficult to assess the prevalence of the disease in the adult, for it is, in the main, a chronic condition and the incapacitated individual may ultimately find his way to the clinic or hospital. In addition, the milder signs of the disease can be picked up in a clinical survey of the affected population. Obviously, if we could relate the incidence of the disease in the adult to its incidence in the infant, an assessment of the health significance of beriberi would be a good deal easier.

There are, however, certain factors which may influence the accuracy of the picture of this relationship. One is a very common, and probably a very important practice in most of the countries concerned, namely, the restriction of the maternal diet for 44 days after the birth of the infant. The woman, already probably on an inadequate intake of thiamine, undergoes the stress of delivery and then subsists for 6 weeks on a diet of rice and dried fish. The lactating woman is therefore a greater risk than other adult members of the community. The recorded incidence in the total population may therefore be misleading.

It may be worth mentioning here that one of the reasons given for the decrease in infantile beriberi in Japan is the improved status of women, whereby they now feed with the men and children and therefore have access to all the foods available to the family instead of only the left-overs (104).

Another is the inconstant relationship between the disease in the mother and in her infant. It might be possible to carry out extensive investigations in lactating women as a means of forecasting the probable occurrence of beriberi in the child. Hirota, however, drew attention to the fact that in some of the first cases of infantile beriberi he described, no signs of the disease were to be found in the mothers, although they developed them some weeks after their infants had died. Bray found little evidence of maternal beriberi, although the disease in the infant was acute and usually fatal. Haridas found signs in only 25% of the mothers of cases admitted to his wards. Fehily drew attention to the absence of any sign of maternal disease in many acute cases in the infant, and suggested that during the pre-clinical stage the mother was likely to lactate well and her child would therefore be more likely to acquire the disease. When she developed the frank disease this would interfere with her lactation, and the child would then be less likely to be affected. It is also recorded that sometimes the mother has beriberi without the child showing any sign of it. It seems difficult, therefore, at the moment to form any opinion as to the relationship of the occurrence of the disease in the mother and her child.

For these reasons there is a need for

further investigations into the whole problem of infantile beriberi and its importance as a public health problem.

One possible approach might be to carry out pilot studies in two similar areas, one in which the rice is highly milled and another where it is hand pounded. The main objective would be to establish the differences in infantile mortality rates, and the population studied would have to be of a sufficient size to allow conclusions to be drawn. In addition to the total births and infant deaths in these two areas, the exact age at death and the mode of death would have to be ascertained. Where possible, of course, sick infants would be seen by a pediatrician. Dietary surveys to establish the degree of imbalance, and clinical surveys of groups of adults are also needed.

After an investigation of this sort, we might be able to establish a relationship between the deficiency of thiamine in the diet and the disease in the adult—possibly with special reference to the lactating woman—and the infantile mortality associated with these more readily measurable entities.

Another approach to this problem might depend on the assessment of the thiamine nutriture of the lactating woman. Some attention is now being paid to the level of thiamine in breast milk. This question has already been discussed today.

A great deal has been known about beriberi for many years and yet this large gap in our knowledge still remains. If we in the international agencies are going to do our job in the prevention of ill health due to beriberi, this gap must be filled.

RAMALINGASWAMI: After the two brilliant expositions this afternoon, it has fallen on me to enter an area of extreme ignorance. There's an old Chinese proverb which says it is much easier to share ignorance than knowledge.

Burgess has just given a review of the existing situation in several countries of southeast Asia with regard to the incidence of infantile beriberi. Let us first go over some of the recognized facts about this condition. Infantile beriberi is seen most commonly in the rice-eating countries, although it has been described among populations not subsisting on rice (94). It is associated frequently with the presence of signs and symptoms of beriberi in the mother. I said frequently because there are quite a few reports to indicate that there need not be any manifestations of beriberi in the mother (91, 108) and in fact it was this observation which led Ito of Japan to postulate in 1911 the 'breast milk intoxication theory' of beriberi in the infant. This theory was later revived by Fehily who pleaded strongly in its favor (100, 109, 110). Breast feeding is the *sine qua non* for the development of infantile beriberi. Professor José Albert of Manila, who had been mentioned by Burgess, writes about this association between breast feeding and infantile beriberi rather forcefully (91). He says, "It is a single fact unparalleled in the history of medicine that human milk as a food appears to be a scourge to the Philippine infant, an inverse phenomenon to the experience of other nations where human milk is the strongest bulwark of child hygiene, the magic liquor that guarantees the life of the infant."

The age of onset of infantile beriberi is of considerable interest. It occurs almost exclusively in the first year of life. It is distinctly uncommon under 1 month of age and is very rare after the first year of life. The period from the first to the fourth months of life is generally regarded as the most dangerous one in which the acute or fulminant (cardiac) forms of infantile beriberi are seen with greatest frequency. This feature is of significance and is made use of in assessing the incidence of infantile beriberi in a community. Aykroyd will probably emphasize this point later. After the fourth month of life, the clinical syndrome tends to be subacute and chronic, although either of these types can revert into the acute fulminant form in the presence of intercurrent factors. The reason for this remarkable pattern of age distribution of infantile beriberi is completely obscure. It has been assumed by some that the child has sufficient reserves of vitamin B_1 in the first month of life (109). There is some indirect support for this in the fact that the maternal requirement for B_1 during late pregnancy is considerably increased (111) and one would like to think that this is

partly to meet the storage requirements of the infant. The vitamin B_1 levels in colostrum and early lactation milk are much lower than those in the late lactation milk (112). Teleologically, this could be interpreted to mean that the infant's requirement for vitamin B_1 in the first 4 weeks of life is less than in the later months.

There are several excellent clinical descriptions of infantile beriberi (91, 94, 100, 108–110). It is customary to divide the syndrome into various types—the acute cardiac or fulminant or cardialgic type, the dry and the wet type, the aphonic type, the pseudo-meningitic type, the peripheral neuritic type, and so on. It is essential to recognize, however, that the syndrome presents a kaleidoscopic picture, with a variable combination of features and that one type frequently merges with the other. In the present state of our knowledge, it seems preferable to conceive of the syndrome in terms of its speed of evolution. Fehily (109) has presented such a concept, which is reproduced in table 7.

Instead of giving a text book description of the syndrome, I should like to comment briefly on some of its prominent features. Of all the forms of infantile beriberi, the one that has attracted the greatest attention is the acute cardiac form. Albert and Ahad (91) give a graphic description of this form which is worthy of repetition. "The baby around three months of age, apparently in good health, nursed entirely by its mother is abruptly seized with an attack of screaming. As he utters his loud piercing cry, his body stretches, the abdomen becomes hard, the pulse thready, the respiration labored, his face either deadly white or cyanotic and an expression of profound terror or suffering grips the entire being. This state may last anywhere from one half to one hour. It disappears spontaneously only to reappear with increasing severity and frequency until death supervenes or specific treatment is given promptly." Treatment with vitamin B_1 administered by any route results in a quick recovery, and I have observed a child who improved remarkably in six hours after intramuscular administration of 25 mg of vitamin B_1. The rapidity of recovery suggests that the lesions are biochemical rather than structural.

Platt referred this morning to vomiting in beriberi. This is a very common sign in infantile beriberi and provides an early indication of the development of infantile beriberi. The aphonia, which Denny-Brown referred to in adult patients with beriberi, is such a striking feature of infantile beriberi that a separate aphonic type has been described. When it is well developed, aphonia leaves a lasting impression on the observer. The child is apparently crying but no sound is heard—the so-called 'visible cry' of infantile beriberi. The aphonia is attributed to either recurrent laryngeal nerve paralysis or to edema of the vocal cords. In a series of 37 cases of infantile beriberi, the majority were found to be due to impairment of movement of the vocal cords (113). The precise etiology of aphonia is not known and requires further study.

The pseudo-meningitis type described by Albert requires further definition. It is characterized by signs and symptoms of involvement of the central nervous system. Strabismus, nystagmus, spasmodic contraction of facial muscles, convulsions and rise of temperature have been described, and it is stated that these cases are often misdiagnosed as either encephalitis or meningitis. The morbid anatomy of this type is unknown. Its relation to Wernicke's syndrome of alcoholics remains to be determined.

The pathological studies of infantile beriberi have contributed little to our understanding of the basic mechanisms in this disease. Miura in Japan (64), Andrews in the Philippines (89), Pekelharing and Winkler (77) in Indonesia, and later Wenckebach in the same area (10), have provided descriptions of autopsy findings in beriberi. This is what Wenckebach says about the heart in infantile beriberi: "The interior of the thorax was a really wonderful sight, the right heart extending greatly to the right, the surface shining, the vessels under the pericardium much engorged and the right ventricle dilated almost to the breaking point. The pulmonary artery was of the same size as the aorta. The heart was as large as that of a normal child ten years old." Details of microscopic features are scanty and all that one gathers is that there is a swelling of the muscle fibers, a kind of

hydropic degeneration. With regard to the other organs, especially the brain, the search in the literature has been unproductive. Intense congestion and edema of the liver, spleen and the brain have been described. Bray recorded fatty changes in the liver, but this is probably coincidental and not directly related to B_1 deficiency. In experimental B_1 deficiency, no fatty changes are found in the liver.

The chemical pathology of infantile beriberi is unknown. The biochemical disturbances in this condition and their relation to the pathological changes and clinical manifestations are obscure. The hypothesis that the syndrome is due to the toxic action of intermediate compounds of carbohydrate metabolism derived from the mother and transmitted through breast milk, is attractive and fits in with the observed facts. However, there is as yet no proof of this. Platt and Lu (5) found methyl glyoxal in the milk, urine and cerebrospinal fluid of adult cases of beriberi, but the role of this substance in inducing infantile beriberi is yet to be determined. Orimo (114) found reduced activity of glyoxalase in the milk of Japanese mothers with beriberi which was corrected on administration of vitamin B_1.

We are just beginning to see some light on the relationship between vitamin B_1 levels in milk and their relation to infantile beriberi. In table 8, I have brought together the pertinent data. All the estimations of B_1 shown in this figure were made using the same method—the thiochrome method. The B_1 levels in the milk of Indian, Burmese and Malay women are markedly lower than those of British and American women. Ramasastri's (116) observations in India were made in an area formerly known as the Northern Circars where beriberi has been known to be endemic for centuries (98, 119). The lowest figures recorded are those of Chitre (118) in a group of women, 50% of whom show evidence of 'beriberi'. Many more observations on B_1 levels in milk, with particular reference to the presence or absence of beriberi in the mothers and infants, are required before the significance of vitamin B_1 levels in milk in relation to beriberi in the mother and child can be ascertained. The findings, however, encourage one to hope that vitamin B_1 levels in human milk in a community may prove to be helpful as indicators of B_1 deficiency. The observations of Sundaranajan (120), which were made in South India, in which he did not find any difference between B_1 levels in milk in women of South India and those of western countries, form an exception to the trend observed so far. He also did not find significant differences in B_1 levels in milk from normal women and women with 'beriberi'. This work has not been presented in the table as the method used by him is somewhat different (112).

TABLE 8. THIAMINE CONTENT OF FULL LACTATION MILK

Country	Year	Thiamine µg/100 ml		Authors
		In health	In beriberi	
U.K.	1944	18.3		Kon and Mawson (112)
U.S.A.	1945	14.8		Roderuck *et al.* (115)
India	1957	12.2		Ramasastri (116)
Malaya	1956	11.3		Simpson and Chow (117)
Burma	1957		8.3*	Chitre (118)

* About 50% of the women in this group showed signs of beriberi.

References have been made in the course of discussion this morning to electrolyte disturbances. Very little attention seems to have been paid to water and electrolyte disturbances in beriberi and experimental thiamine deficiency. We know that during the period of recovery of the nerve after the passage of an impulse the sodium that has entered the cell is extruded into the extracellular space and the potassium re-enters the cell; this is an active process requiring energy. Since the oxidative mechanism of the nerve cell and the cardiac muscle cell in thiamine deficiency is disturbed, it is possible that there is continual depletion of potassium from nerve cells and maybe heart cells. Pecora (61) finds decreased potassium and increased sodium levels in cardiac and skeletal muscles in thiamine-deficient rats and believes that these disturbances account partly for a cardiac damage in beriberi.

The edema in beriberi has never been explained satisfactorily. Guggenheim found recently that thiamine-deficient animals have impaired ability to inactivate Pitressin

and I'd like to recall here some observations which Sir Robert McCarrison made in the course of his work on experimental beriberi 40 years ago indicating the possible significance of the adrenal gland in the genesis of edema in beriberi. In 1919 McCarrison wrote as follows: "The most remarkable result emerging from this research is the demonstration of hypertrophy of adrenals which results from a dietary deficiency in the antiberiberi vitamins". He used pigeons, and he goes on to say that "in the autopsied animals nothing is more remarkable than the appearance of the atrophic testicles and the neighboring hypertrophic adrenals. In other healthy birds the large testicles are 50 times the size of the adrenals whereas in the diseased birds the latter may actually be larger than the former" and finally concludes, "I'm led therefore to believe that an intimate and causal connection exists between the atrophy of adrenals and the origin of edema." Forty years later, we still don't know what part the suprarenal cortex plays in the edema of beriberi. There are many gaps in our knowledge of infantile beriberi. I should like to conclude this very inadequate and unsatisfying review with the comment that beriberi is a problem in geographic pathology and, in the words of Blackburn, "in the search for causation of disease, the study of geographic pathology is of the greatest importance," and I hope something will come out of this meeting which will fill many of the gaps that exist today.

AYKROYD: I have, sir, only a couple of points to add to these two most interesting accounts of infant beriberi. One is this: I personally believe that a peak in infant mortality between the ages of 2 and 5 months of life is an unique phenomenon in public health statistics and that whenever you can establsh such a peak you can assume that there is infantile beriberi in the population. I have consulted public health statisticians on this point and they, after scrutinizing the literature of the subject around the world, have told me that as far as the existing infantile mortality statistics are concerned, there is no other cause which can produce a picture of infant mortality such as that produced by infantile beriberi. That may possibly simplify the approach to the problem of discovering whether, in a given area, you have infantile beriberi, although in many of the areas in which its existence might be suspected, detailed figures indicating the 'timing' of mortality in the first 6 months of life are lacking. Another interesting point are the differences reported in the proportion of women whose infants develop beriberi and who themselves show signs of thiamine deficiency. No explanation of this is at present forthcoming. Some workers have put the proportion at 50% or more, while others have reported mothers of infants with beriberi are themselves usually free from obvious signs of the disease.

Ramalingaswami has remarked that the treatment of an acute case of beriberi in an infant with thiamine is one of the most dramatic things in medicine. He mentions 6 hours as a period in which the infant showed definite improvement. I myself have seen a case in which I could have sworn that the child, moribund, seemed obviously better within 2 hours. I suggest that this condition in infants is as near as one can get in human beings to a pure thiamine deficiency. It does closely resemble in certain respects the claimed pedina beriberi in adults as this appears in the textbooks. I personally prefer to call the condition in infants 'thiamine deficiency'.

MCLAREN: I'd like to make two suggestions for need for further work, one in the experimental field and one in public health. Firstly, there is evidently need for studies in experimental animals simulating the conditions under which infantile beriberi occurs. The second point is that there do not seem to be any studies on the volume of milk produced by lactating women in areas where infantile beriberi is common. We have a number of values of the concentration of thiamine in milk, but I think it is evident, particularly from work with regard to protein malnutrition, that the volume of milk, as well as the concentration of nutrients, must be taken into account.

HANDLER: Of perhaps equal importance would be an analysis of the change, if any, in the fat and carbohydrate contents of milk. It wouldn't take a very large reduction of fat to convert milk to a high carbohydrate foodstuff; if the thiamine content

is reduced by half and the fat lowered at the same time, the nutritive properties are, of course, greatly altered.

PATWARDHAN: I might add to what Ramalingaswami said about the determinations of thiamine in human breast milk carried out by one of my colleagues. These were done in an area in which endemic beriberi is believed to be present. I must correct Ramalingaswami to that extent because cases of infantile beriberi were reported by Aykroyd and Krishnan (98) about 13 years ago almost next door to the area concerned. The results which you saw in the chart are from this area, though I must admit that today frank cases of beriberi among adults have been found to be relatively few. For this, we have no information on the current incidence of infantile beriberi in that particular region. Ramasastin has analyzed samples of milk in certain localities within the region where the values for thiamine have been very much lower, of the order of 7 or 8 mg/100 ml. We have recently analyzed 108 samples of human breast milk during different periods of lactation. Once the milk matures at the end of 5 weeks there is little change in the thiamine content; between 14.5 to 16.5 mg/100 ml have been obtained in common samples, which is not very much different from the average values which have been reported in well-nourished mothers. The values of 12 mg/100 ml which are apparently lower are from an area where we have reason to believe that beriberi is still present in spite of our inability to detect frank cases of beriberi in an appreciable number of people within the area.

Now the question with regard to the whole output of milk of malnourished mothers which McLaren has asked. In our investigations on protein malnutrition my laboratory has carried out several studies by a test feeding technique in infants and children up to 2 years of age; we have found that in the first 6 months the total output of milk, in spite of malnutrition, was not greatly inferior to what one would expect from well-nourished mothers. Further experiments at Coonoor have shown that supplementing these mothers with additional protein did not materially increase the output of milk. The point which Handler raised also can be answered from the results of analysis of milk which we have done so far. At one time, it was mentioned in relation to kwashiorkor that the protein-fat ratio in the milk of African malnourished mothers was low. I do not think that this finding has been substantiated by further work either in Africa or elsewhere. We have found that the proportion of fat, protein and carbohydrate in breast milk of Indian malnourished women is almost the same that one would obtain in the milk of well-nourished mothers. Unfortunately, we had no information on the incidence of infantile beriberi in the region in which we are now working. Also we had no information on the incidence of beriberi in the infants or the siblings of those mothers in whom we particularly found low values of thiamine. Finally, I would say that with regard to the major constituents of milk, there is practically no difference as compared to the constituents of milk obtained in well-nourished mothers, and if one were to look for a relationship between breast milk and beriberi, it must be sought principally in the quantity of thiamine, which is contained in this form, although I daresay that this concept may not be applicable primarily to all areas where it has been so endemic.

BURGESS: May I point out that the child who gets beriberi is often the well-nourished child, most frequently, the pride and joy of the household, the baby boy.

SCRIMSHAW: Ramalingaswami noted that there are resemblances between infantile beriberi and kwashiorkor. Since he is personally familiar with the clinical manifestations of both infantile beriberi and kwashiorkor, I hope he will indicate whether he thinks this suggestion to be misleading. Certainly the time of occurrence of beriberi early in the first year rather than after weaning is strikingly different from kwashiorkor, as is its development in children who are receiving adequate mother's milk. Certainly kwashiorkor wouldn't respond in 2 or 6 hours to thiamine administration.

RAMALINGASWAMI: I only mentioned this just to indicate the fatty changes in the liver; experimental thiamine deficiency in rats doesn't lead to any morphological lesions in the liver and it is possible that there are other deficiencies associated with this. I didn't think that there were. I'm not

suggesting that infantile beriberi and kwashiorkor are one and the same thing.

SCRIMSHAW: I doubt if it's fair to imply that the fatty liver of beriberi is the protein deficiency type seen in kwashiorkor, if infantile beriberi develops in children who receive adequate quantities of breast milk for their protein needs, and as Burgess pointed out, are the "pride and joy of the household."

C. *Rice*

AYKROYD: Mr. Chairman, we have moved through many different worlds in the course of today's discussions. Beriberi is largely a rice-eaters disease, as you pointed out in your opening statement this morning. Study of the relationship between milled rice and beriberi played a crucial part in the discovery of the vitamins. We know, of course, that beriberi can occur on a staple of refined wheat flour, as in Newfoundland, but by and large it is a rice-eater's disease. Rice is the most important cereal in the world, quantitatively speaking, being the staple food of more than half the human race. Ninety per cent of total world rice production is in Asia. I had the pleasure the other day of talking to the American Rice Miller's Association in Galveston, Texas—a very vigorous group. I think that they were a little bit taken aback when I told them that only 2% of the total world production of rice is in the U. S. A.! You have to eat a great deal of rice to get beriberi; that is, the percentage of calories from rice must be high.

The way to prevent beriberi is to increase the intake of thiamine. There are a number of different ways of doing this. In the first place, there is the general improvement of the diet, so that it contains less rice and more of other foods. Such a change is unquestionably happening in many parts of the world in which beriberi was once a serious problem. Japan affords an interesting example, and there are also other parts of South and East Asia which could be mentioned. In Japan, for example, particularly since the war, there has been very considerable increase in the intake of wheat because of rice shortage; the same has happened elsewhere. Of course, one would not expect that the substitution of milled rice by refined wheat flour would have a preventive effect. In Japan, as I shall mention later, the widespread use of thiamine has also had an important effect on the prevention of beriberi.

The other obvious method of getting more thiamine into the diet is a change from milled rice to rice which has a higher thiamine content. In early days, after workers in Java and Malaya and elsewhere had demonstrated the relationship between milled rice and beriberi, it was natural to stress, as a practical policy, the encouragement of the use of either home-pounded or under-milled rice. If you read the Proceedings of the meetings of the Far Eastern Congress of Tropical Medicine during the first two decades of this century, you will see that they kept insisting on this point, emphasizing either the preservation of the habit of home-pounding or the use of under milled rice, instead of highly milled rice. You can sum up the results of all those efforts by the simple statement that they have been ineffective except in some rather rare instances.

It has in fact proved extremely difficult to encourage the use of under-milled rice or to preserve the practice of home-pounding. In this connection, it should be borne in mind that home-pounding rice is a tedious chore. In ancient Rome, household slaves who had misbehaved were sometimes punished by being set to grind wheat in the heavy stone querns of antiquity. The chore of home-pounding rice is not quite so laborious, but nevertheless people will always avoid it when they can; if there is a machine to do the job for them they will use the machine. I regard attempts to restrict or to prevent the spread of rice mills as impracticable as a practical policy. As things are, the number of people who pound rice by hand or by foot in the world diminishes steadily every year.

Another method of approach would be to replace highly milled raw rice by milled parboiled rice. About one-fifth of the whole world's rice crop is parboiled. Burgess mentioned this morning that, as long ago as 1907, Braddon demonstrated in Malaya that those who eat parboiled rice do not get beriberi. He himself thought that the steaming which is part of the parboiling

process destroyed a toxin in the rice. There are various reasons why parboiled rice is preventive of beriberi. Generally speaking, it is eaten in a less highly milled state than raw rice. More important is the fact that the steaming causes a change in the position of the thiamine in the grain, so that it is not removed when the milling process takes place, and equally it is more difficult to wash it out of parboiled rice than out of raw rice. It is perfectly possible to produce a milled parboiled rice of good commercial quality without using the elaborate 'rice conversion' process which is popular in this country. There are various ways in which the ordinary parboiling process could be improved.

The use of parboiled rice is largely confined to Indians and descendants of Indians in different parts of the world. A change-over from raw rice to parboiled rice is difficult to effect because of the conservatism of rice eaters and their preference for a certain kind of rice. There have been one or two successful attempts, here and there, to introduce parboiled rice in place of raw milled rice, but by and large, the method of increasing thiamine intake has not been effective. Perhaps this is because enough effort has not been made.

Another method of increasing thiamine intake from rice is to reduce the amount of washing to which the rice is subjected before consumption. Platt has shown that when Chinese factory workers got rice which was not washed the incidence of edema and beriberi decreased. Those observations on human beings have been fully supported by laboratory experiments indicating that a high percentage of thiamine is washed out of raw rice by the ordinary household washing procedures followed in many parts of Asia. Incidentally, if you are trying to produce acute thiamine deficiency in pigeons, you can hurry up the process by giving them rice subjected to very vigorous washing; this is sometimes useful when you want to demonstrate the condition to students. You can reduce the time it takes to induce 'head retraction' from about 5 or 6 weeks to 3 weeks. Here again, I do not think that we should attempt to change household habits with respect to the washing of rice. Most rice sold in rice-eating areas requires washing before consumption to remove dirt and other impurities. The effect of cooking is less important.

Another interesting approach is that of rice enrichment. The method of enrichment is familiar in general to nutrition workers. Unquestionably rice enrichment enriches rice; it increases the thiamine content of rice as consumed and populations which are prone to beriberi would escape it if they switched over from milled rice to enriched milled rice. There is no question about that; from this point of view the approach is lucid and sensible. Unfortunately, in practice rice enrichment is unlikely to be successful, except in limited areas and under special circumstances. It is particularly difficult to introduce or organize when there are large numbers of small rice mills which need careful inspection and control, and these are the areas in which the problem of beriberi appears to be now growing. Finally, reference may be made to the Japanese experience with respect to beriberi. Burgess and I were in Japan in 1956 attending a nutrition committee meeting and we were extremely interested in the Japanese approach. There was a great deal of beriberi in Japan 30 or 40 years ago. Now there is very little. They can produce some cases here and there, but it is not a public health problem of any importance. To some degree this is the result of the general improvement in the diet to which I have referred, but it is also to a considerable extent the result of extensive production and distribution of synthetic thiamine. Japan produces large quantities of thiamine, which can be sold at very low cost, and which reaches the population widely. In Japan the people are familiar with beriberi and with the effect of thiamine in prevention. Its widespread use has played an important part in the prevention of the disease in that country. I personally believe that the greater use of synthetic thiamine in a variety of ways, including its distribution to pregnant women and lactating women, would be one of the most effective methods of preventing beriberi, particularly infantile beriberi which is the most important problem in this field at the present time. In conclusion, I feel that our efforts to solve the beriberi problem have been somewhat disappointing. As Burgess

pointed out, what we specially need now are detailed field studies of the incidence of beriberi, particularly in infants, which would demonstrate clearly to governments and public health authorities that the problem exists, thereby prompting them to take action and to initiate effective campaigns to prevent the disease.

VII. The Pathogenesis of Beriberi

Follis: This part of our program was designed to deal with the pathogenesis of beriberi, that is, how the various clinical and pathological manifestations of the disease come about. Before going into this subject, it may be worthwhile to say a few words on the pathogenesis of deficiency disease in general. Note that I use the term, deficiency disease, and not nutritional disease. I think there's coming a time when we may have to stop using the term nutritional disease to refer to deficiency states, particularly because in this country we're not nearly as concerned with undernutritional disease as we are with overnutritional disease. The development of a deficiency state has a fairly definite progression. Figure 14 depicts an approach which was developed by Dann and Darby (105) and has been helpful to others for a number of years. Decreased intake of a nutrient or the effects of certain conditioning factors may lead to lowered concentrations of the particular material in the plasma, which in turn leads to lowered concentrations in the intercellular fluid or in the cells, of which one may readily study erythrocytes or white blood cells. Such changes may in turn be reflected by alterations in intracellular concentration of the nutrient. One may then expect to find biochemical and physiological alterations. Finally, morphologic changes appear and these are followed by death of the cell.

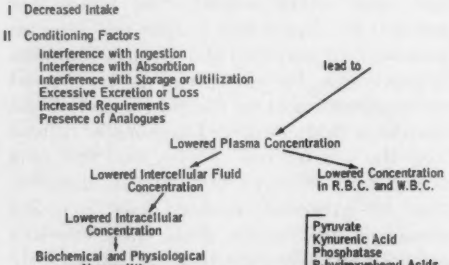


Fig. 14. Pathogenesis of deficiency disease. See (31).

This approach applies to deficiencies of exogenous nutrients. We also are becoming aware of a host of syndromes which arise from deficiencies of endogenous nutrients, that is, materials which do not necessarily have to be in the diet but which are made by the organism itself. The integrity of cells and tissues is severely affected if such materials are deficient. The hypoglycemic syndrome is one example of this (31).

Next, I want to make another point which is important. If you look at the history of the development of our knowledge of nutrition since the very beginnings, you will find that a fairly consistent pattern has been followed (106). A clinical syndrome such as scurvy, beriberi, rickets or pellagra was described; then its pathologic manifestations were delineated. Sometimes, as in the case of scurvy, a preventive material was discovered; in other instances, and more usually, a similar disease syndrome was produced in experimental animals. As a result, some preventive material could be isolated, identified and synthesized. Modern biochemistry, particularly intermediary metabolism, has benefited enormously from all this; yet during the course of such development, study of those diseases that started everything has largely been neglected. Consequently, I hope that this conference may help us return to basic studies of the syndromes which have led to many of the advances in present-day biochemistry.

Finally, it is also worthwhile to point out, as is well recognized by many but perhaps not by all, that a number of deficiency syndromes result from a lack of multiple factors. This is likely true of beriberi. I continue to feel that the terms thiamine deficiency and beriberi are not synonomous. Rice is deficient in other nutrients besides thiamine.

TABLE 9. THE PATHOGENESIS OF BERIBERI

Host	Versus	Environment
I. Racial		I. Rice
II. Nutritional status at birth		(1) Production: water, fertilizer, amount of land, seasons, etc.
III. Social factors, especially weaning		(2) Processing: milling procedures
IV. Infections		(3) Preparation in home
V. Anemia		(4) Nutritive properties
VI. Diarrhea		(a) Amount (calories)
VII. Pregnancy-lactation		(b) Vitamin content
VIII. Growth and metabolic activity of tissues		(c) Mineral content
IX. Specific response with respect to:		(d) Lipid content
(1) Heart		(e) Quantity and quality
(2) Nervous tissues		II. Other food supplements
(3) Edema		III. Animate agents of disease
		IV. Miscellaneous
		(a) Toxic factors

Now for the pathogenesis of beriberi. Disease is the reaction of the host to some abnormality or abnormalities in its environment. In the case of beriberi the host is man; the main factor in his environment is rice. Hence, in its simplest terms beriberi reflects the results of the interaction between man and rice. This is, of course, a very simple way to define a most complex state of affairs.

First, let us consider rice. Some of the important points with respect to this cereal grain are summarized in table 9. We must deal with rice production; here, the amount of available land and water as well as the adequacy of fertilizer will affect the supply of rice. Here, too, perhaps the genetics of rice enters the picture, since certain types of the grain have different protein or vitamin contents. We've already heard how processing or milling procedures affect the nutritive property of rice. A this point the host begins to play a role, since he prefers, for various reasons, polished grain rather than that which is partially hulled. Finally, the mode of preparation of rice in the home must be considered; such include washing procedures before and after cooking, boiling time, etc.

All of these factors affect the nutritive properties of the grain. The amount available per day affects the total calorie intake as well as the amounts of vitamins, lipids, minerals and protein ingested. Of the water-soluble vitamins, thiamine is of great importance. Besides being low in essential fatty acids, polished rice is deficient in vitamins A and D. Further, it lacks sodium, chloride and calcium; the calcium-phosphorus ratio is abnormal. Finally, the quantity of protein is of importance; its quality has already received our attention. How other foodstuffs supplement the diet must also be considered; according to WHO-FAO reports the materials which commonly supplement the rice eater's diet are small amounts of fish, vegetables, pulses and fruits.

Besides the host's nutritional environment one must consider other aspects of the world about him. In every tropical and subtropical area, animate agents of disease: bacteria, fungi, parasites, have to be given much attention, since they may enhance or precipitate deficiency disease in the host. The presence of certain toxic factors, spoilage of food, etc., must always be borne in mind. So, too, heat, humidity and other physical factors must be taken into account. This, then, is just a brief resumé of the complexity of the environment which makes comparable studies in the cloistered laboratory so difficult.

When we come to consider the host, the problem immediately arises as to whether there is any racial disposition to beriberi. There is no evidence for this, though such might be inferred by the differences in response to deficiency states in various animal species. The state of nutrition at birth immediately becomes important. Individuals born into areas where beriberi has been endemic for centuries have a heritage of undernutrition. The milk of their deficient mother is probably poor in quality

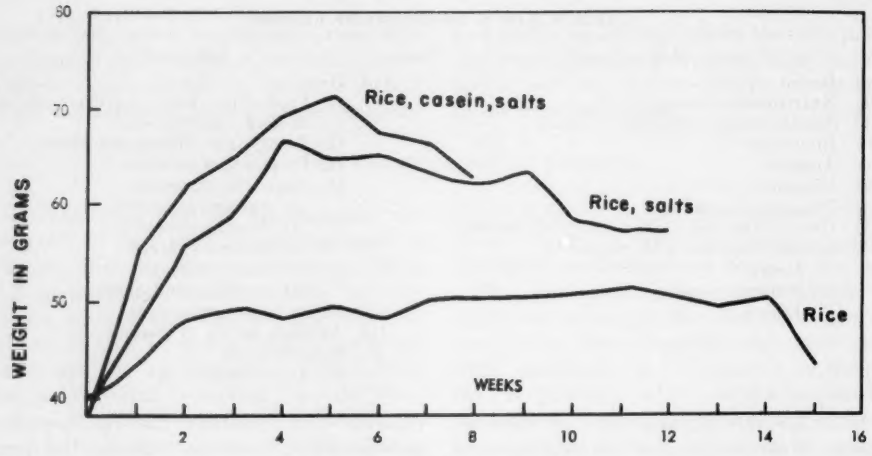


FIG. 15. See text for explanation.

as well as in quantity. Furthermore, social factors, such as weaning habits, are of extraordinary importance. For instance, in many areas the infant is weaned to a diet of rice, which the mothers may chew up and place in its mouth or which may be fed as a gruel. There may be little supplementation of the diet in childhood.

The host may harbor many of the animate agents of disease which are prevalent in the environment. The possible role of intestinal bacteria in destroying thiamine has been mentioned. Intestinal infections lead to diarrhea, which may precipitate deficiency states. Moreover, anemia commonly accompanies parasitic infestations. In adult females, multiple pregnancies deplete them and their milk of stores of essential nutrients.

Finally, a most important consideration is the state of metabolism and of the metabolic balance of the organism. There would appear to be a fundamental law which states that when imbalance or insufficiency are present a basal state is preferable to no state at all. This can best be illustrated by figure 15. *Curve A* refers to a group of rats weighing 40 gm which was placed on a diet of ground polished rice; such animals gain a little, but only about 10 gm. Their weight curve then plateaus and finally drops with death after 16 weeks. In *curve B*, 5% salt mixture has been added to the rice diet. Growth is better but the animals don't live as long. In *curve C*, casein was added to the salt mixture; here growth is even better but the animals die twice as soon as those on the basal rice diet alone. It seems to me that one sees this phenomenon time and time again in nutritional studies. It illustrates, as we have already noted, a principle: if the metabolism is raised above a certain basal level in the presence of deficiencies or imbalances such an effect will be deleterious, since stores of essentials will be used up on this higher plane of metabolism. Moreover, at such a higher level, growth and metabolism of certain cells can take place, albeit abnormally, hence biochemical and anatomical alterations may make their appearance. For instance, there must be growth of epithelium to have certain of the skin lesions of pellagra or kwashiorkor develop. Moreover, if one starves an experimental animal skin lesions aren't seen; however, with single deficiencies of the B-group very definite and different lesions do appear. A similar situation doubtless operates with respect to beriberi. There must be growth and metabolism in order to have lesions appear. Moreover, the degree of deficiency is of importance, as has already been mentioned today. This concept is all beautifully illustrated by the 'INCAP pyramid' of marasmus versus kwashiorkor (107). In marasmus there is growth retardation and weight loss, so that there is little

chance to develop the skin changes, fatty liver and other manifestations of kwashiorkor.

This brief resumé brings us to try to account for the cardiac, central and peripheral nerve lesions, as well as the edema, which form the basis for the clinical and pathologic manifestations of beriberi. We have heard from Handler how thiamine functions in biochemical reactions. How this leads to myocardial necroses, to the lesions in peripheral and central nervous tissues and to edema, remain unanswered questions. Moreover, since rice is deficient in other nutrients and since we have already stated that beriberi and thiamine deficiency are not synonomous, the role of other essentials in leading to the beriberi syndrome have as yet not been delineated.

This very brief summary can only indicate the complex nature of the problems. The factors which have to do with rice as well as the host are many and complicated. Yet beriberi is no exception, for this is the situation which confronts one in the analysis of any disease.

HIGGINSON: I would just like to have one point settled. Is the term 'beriberi,' as used by the majority of people here, applied to any nutritional condition in which thiamine deficiency is believed to be the major etiological factor, that is, whether it is conditioned by alcohol or not? There appears to be a certain spectrum in the usage of the term, for example, the difference Oriental and Occidental beriberi.

OLSON: On this question of what represents the syndrome of beriberi, I would hesitate to agree that thiamine deficiency seen in alcoholics is not beriberi merely because it occurs in the United States. Although we are bound to a certain extent by the traditional definition of a clinical syndrome in deciding what is and what is not beriberi, it must represent a portion of the spectrum of thiamine deficiency in man, no matter where it occurs. The width of this spectrum extends from the extremely severe state of thiamine deficiency represented by Wernicke's encephalopathy to extremely mild states which, in America, are probably represented by peristent chronic nonremitting peripheral neuropathies in alcoholics. Somewhere in the middle of this gradient between these two extremes we have both 'wet' and 'dry' beriberi. To say, on the other hand, that there is a sharp distinction between alcoholic beriberi and Oriental beriberi, I think would be unjustified. Further, as Oomen has pointed out, some Indonesians drink alcohol and it accelerates their beriberi.

PATWARDHAN: I'm conscious that I'll probably stir a hornet's nest when I question the validity of the so-called established relationship between thiamine to non-fat calories ratio and beriberi. It has been accepted that, even in humans, states of thiamine deficiency could be predicted from the ratio of thiamine to non-fat calories in the diet. But I am somewhat doubtful of this because of our experience, which has shown that, in spite of this ratio being below the accepted level, there has been hardly any incidence of beriberi or disturbance in the ratio of pyruvic acid to lactic acid. I feel that the validity of this ratio as an indication of nutritional status with respect to thiamine and susceptibility to beriberi requires to be freshly evaluated in human nutrition.

My second point, which relates to the pathogenesis, and which is probably of importance in relation to other deficiency diseases as well, is, what are the factors in thiamine deficiency which would lead to wet beriberi in one case, if I may use that term, and dry beriberi in another. Do we know enough about that? Can we reproduce the conditions experimentally at will? I think this is one area in which our understanding of the influence of deficiency of a vitamin leading to a particular clinical manifestation of one type or the other (where there are more than such manifestations) is extremely deficient and we ought to improve it. Thirdly, I would like to go back to the important question which I asked this morning. What are the observations which would be helpful in assessing the incidence of subclinical beriberi or thiamine deficiency in population groups? It is extremely important that field surveys should be carried out in order that an incidence of beriberi could be ascertained in those areas where the problem appears to be assuming greater importance. I do not

think that this particular topic has been discussed at all today.

HANDLER: Before abandoning the notion of a relationship between non-fat calories and the thiamine intake, there is one other possibility which may be involved in infantile beriberi, which now seems most mysterious to me. That is the possibility of a positive etiological agent in the milk. Thus, a load of methyl glyoxal imposed on a partial thiamine deficiency might do damage. For the assay which Patwardhan seeks, I might suggest the rate of respiration of erythrocytes in the presence of methylene blue. The direct oxidative pathway is dependent on thiamine pyrophosphate, and is accelerated by the presence of methylene blue. This is a fairly easy measurement and might perhaps be useful. We should not leave the subject of thiamine deficiency without noting that thiamine deficiency is a genetic disease of man. Presumably we are descended from ancestors who could make thiamine, and one of whom lost the ability to do so. Knowing nothing of the biogenesis of thiamine in those organisms capable of such synthesis, we don't know at what point the block lies. However, that there must be such a block is self-evident. We now know where the block is in ascorbic acid synthesis and such information may prove of value in thiamine deficiency. Since Follis mentioned the genetics of rice, it seems to me we should note that we cannot make thiamine, although our ancestors presumably could.

SCHAEFER: I have a comment in reference to Patwardhan's point on subclinical nutrition. The discussions today have repeatedly referred to tools for diagnosis, yet I failed to hear any mention as to the value, if any, of urinary excretion levels. I personally believe that urinary thiamine is a most useful tool for the assessment of subclinical thiamine nutriture.

DARBY: It seems to me that it would be very useful to get some succinct thoughts concerning the usefulness of three types of information which might bear on the appraisal of thiamine status: dietary, including significance of such calculations as the thiamine-non-fat ratio; physical findings, especially the usefulness of readily detected neurologic changes such as the loss of reflexes with or without other findings; and, finally, the several types of biochemical information which may be obtained. It would be useful to indicate which of these might be helpful in studies in population groups.

MACKENZIE: The other side of this that I would like to see taken up is what experiments might be designed in the laboratory to throw more light on the primary biochemical lesion and the succeeding biochemical lesions that manifest themselves in the cellular changes, particularly with respect to the nerves, and the heart, if this be different, though I have heard nothing to indicate that the heart lesions may not be manifestations of changes in functions in the nerves. It seems to me that one of the purposes of this conference was to stimulate experimental pathologists to look for answers, not only in the field but in the laboratory, that might explain the pathogenesis of disease in the field which is very difficult to control and, I am afraid never gives a definitive answer to the problem.

HIGGINSON: I would just like to say a definite effort should be made to study more of the pathology of these conditions in man and that some effort should be devoted to that purpose.

OOMEN: Handler talked about beriberi as a genetically conditioned disease, but I want to stress the point that it is essentially a man-made disease, a disease of the beginnings of the industrial age in the rice-eating countries.

BURGESS: Gentlemen, I shall not attempt to summarize today's most interesting discussions. In view of the lateness of the hour, I shall reluctantly have to adjourn this session. We shall have a chance to consider beriberi with respect to research and training at another time.

REFERENCES

1. BRADDON, W. L. *The Cause and Prevention of Beriberi*. London: 1907.
2. WRIGHT, H. *Stud. Int. Med. Res. F.M.S.* 2: 1, 1902.
3. LU, G. D. AND B. S. PLATT. *Trans. Ninth Congress, Far East Assoc. Trop. Med., Nanking*, 1: 379, 1934.
4. PLATT, B. S. AND G. D. LU. *Quart. J. Med. N.S.* 5: 355, 1936.
5. PLATT, B. S. AND G. D. LU. *Biochem. J.* 33: 1525, 1939.

6. King, T. and L. T. Ride. *J. Obst. and Gynec. Brit. Emp.* 52: 130, 1954.
7. Platt, B. S. *Trans. Roy. Soc. Trop. Med. & Hyg.* 31: 493, 1938.
8. Platt, B. S. and R. Alley. Chinese Medical Association, Shanghai, China, 1937.
9. Lim, E. Q. *J. Hong Kong Univ. Med. Soc.* 17: 215, 1938.
10. Tull, J. C. Summary in English of *The Beriberi Heart* by K. F. Wenckebach. Singapore: Govt. Print. Off., 1935.
11. Aalsmeer, W. C. and K. F. Wenckebach. *Herz und Kreislauf bei der Beriberi*, Berlin: 1938.
12. Platt, B. S. and E. F. Yang. Proc. Physiol. Sect. Third Gen. Conf. Chin. Med. Assoc., Canton, 1935.
13. Platt, B. S. In: *Handbook of Tropical Dermatology*, by R. D. G. Simons. London: 1953.
14. Heard, C. R. C. and B. S. Platt. Proc. Nutr. Soc., 1958.
15. Hawes, R. B., E. S. Manteiro and C. E. Smith. *J. Malayan Br. Brit. Med. Assoc.* 1: 87, 1937.
16. de Langen, C. D. and A. Lichtenstein. *A Clinical Text Book of Tropical Medicine.* Amsterdam: 1936.
17. Peters, J. P. *Body Water.* London: 1935.
18. Thompson, R. H. S. *Biochem. J.* 28: 909, 1934.
19. Platt, B. S. and G. D. Lu. Proc. Third Gen. Conf. Chin. Med. Assoc. Canton, 1935.
20. Platt, B. S. *Brit. Med. Bull.* 12: 78, 1956.
21. Simon, L. J. and L. Piaux. *Bull. soc. chim. biol. Paris* 6: 477, 1924.
22. Platt, B. S. *Trans. Roy. Soc. Trop. Med. and Hyg.* 52: 189, 1958.
23. Johnson, R. E. and H. T. Edwards. *J. Biol. Chem.* 118: 427, 1937.
24. Lu, G. D. and B. S. Platt. *Biochem. J.* 33: 1538, 1939.
25. Aalsmeer, W. C. *Klin. Wschr.* 11: 362, 1932.
26. Victor, M. *J.A.M.A.* 167: 65, 1958.
27. Peters, R. A. *Brit. Med. Bull.* 9: 116, 1953.
28. Platt, B. S. *Ann. soc. belge méd. Tropicale.* In press.
29. Heard, C. R. C., B. S. Platt and R. J. C. Stewart. *Proc. Nutrition Soc.* In press.
30. Dürck, H. *Untersuchungen uben der pathologische Anatomie der Beriberi.* Jena: 1908.
31. Follis, R. H., Jr. *Deficiency Disease.* Springfield: Thomas, 1958.
32. Payet, M., E. Berta, P. Pene and R. Camain. *Bull. Soc. Path.* 46: 688, 1953.
33. Bedford, E. D. and G. L. Konstam. *Brit. Heart J.* 8: 236, 1946.
34. Davies, J. N. P. *East Afr. M. J.* 28: 10, 1948.
35. Ball, J. D., A. W. Williams and J. N. P. Davies. *Lancet* 1: 1049, 1954.
36. Gillanders, A. D. *Brit. Heart J.* 13: 177, 1951.
37. Higginson, J., A. D. Gillanders and J. F. Murray. *Brit. Heart J.* 14: 213, 1952.
38. Gelfand, M. Personal communication.
39. Grusin, H. *Circulation* 16: 27, 1957.
40. Keely, K. J. Personal communication.
41. Grusin, H. *Circulation* 9: 860, 1954.
42. Griffith, R. L. *Arch. Int. Med.* 89: 743, 1952.
43. Blankenhorn, M. A. *Ann. Int. Med.* 23: 398, 1945.
44. Jaffe, R., B. Gavaler and C. A. Domenguez. *Arch. Venezal. Pat. Trop.* 2: 183, 1954.
45. North, J. D. K. and H. M. Sinclair. *Arch. Path.* 62: 341, 1956.
46. Williams, R. D., H. L. Mason, B. F. Smith and R. M. Wilde. *Arch. Int. Med.* 69: 721, 1942.
47. Williams, R. D., H. L. Mason, M. H. Power and R. M. Wilde. *Arch. Int. Med.* 71: 38, 1943.
48. Olson, R. E., O. H. Pearson, O. N. Muller and F. J. Stone. *J. Biol. Chem.* 175: 489, 1948.
49. Hackel, D. B., W. T. Goodale and J. Kleinerman. *Am. Heart J.* 46: 883, 1953.
50. Burwell, C. S. and L. Dexter. *Trans. Assoc. Am. Phys.* 60: 49, 1947.
51. Olson, R. E. and W. R. Schwartz. *Medicine* 30: 21, 1951.
52. Ashburn, L. L. and J. V. Lowry. *Arch. Path.* 37: 27, 1944.
53. King, W. D. and W. H. Sebrell. *Pub. Health Rep.* 61: 410, 1946.
54. Hundley, J. M., L. L. Ashburn and W. H. Sebrell. *Am. J. Physiol.* 144: 404, 1945.
55. Hundley, J. M. and W. H. Sebrell. *Pub. Health Rep.* 61: 847, 1946.
56. Pecora, L. J. and J. M. Hundley. Unpublished results.
57. Rosenberg, H. R. and R. J. Culek. *J. Nutrition* 63: 477, 1957.
58. Pecora, L. J. and J. M. Hundley. *J. Nutrition* 44: 101, 1941.
59. Pecora, L. J. *J. Nutrition* 49: 621, 1953.
60. Hundley, J. M., H. R. Sandstead, A. G. Sampson and G. D. Whedon. *Am. J. Clin. Nutrition* 5: 316, 1957.
61. Pecora, L. J. *Am. J. Physiol.* 169: 554, 1952.
62. Matsukawa, D., S. Chang, M. Fuzemiya, K. Takalo and Y. Harikawa. *J. Vitaminol.* (*Jap.*) 2: 1, 1956.
63. Denny-Brown, D. *Medicine* 26: 41, 1947.
64. Miura, K. *Beriberi.* Suppl. Bd. Nothnagel's *Spez. Pathologie und Therapie*, Wien, 1913.
65. Shattuck, G. C. *Am. J. Trop. Med.* 8: 539, 1928.
66. Strauss, M. *Am. J. Med. Sc.* 189: 378–382, 1935.
67. Strauss, M. *Proc. Assoc. Res. Nerv. Ment. Dis.* 21: 141, 1943.
68. Peters, R. A., L. S. Stocken and R. H. S. Thompson. *Nature* 156: 616, 1945.
69. Kagawa, S. *J. Med. Sci.* (*Sect. VIII*) 5: 1, 17, 43, 1938.
70. Victor, M. and R. D. Adams. *Proc. Assoc. Res. Nerv. Ment. Dis.* 32: 526, 1953.
71. Denny-Brown, D. and J. M. Foley. *Neurol.* 5: 510, 1955.
72. Joliffe, N., H. Wortis and H. D. Fein. *Arch. Neurol. Psychiat.* 46: 569, 1941.
73. Phillips, G. B., M. Victor, R. D. Adams and

C. S. DAVIDSON. *J. Clin. Invest.* 31: 859, 1952.
74. SWANK, R. L. *J. Exper. Med.* 71: 683, 1940.
75. MEYER, A. *Brain* 24: 47, 1901.
76. CARMICHAEL, E. A. AND R. O. STERN. *Brain* 54: 189, 1931.
77. PEKELHARING, C. A. AND C. WINKLER. *Beriberi: Researches Concerning its Nature and Cause, and the Means of its Arrest* (English trans. by J. Cantlie). London: 1893.
78. DENNY-BROWN, D. AND D. BRENNER. *Arch. Neurol. Psychiat.* 51: 1, 1944.
79. FOLLIS, R. H., JR. AND M. M. WINTROBE. *J. Exper. Med.* 81: 539, 1945.
80. ALEXANDER, W. F. In: *Vitamin B_{12} and Intrinsic Factor. 1. Europaisches Symposion, Hamburg, 1956*, edited by H. C. Heinrich. Stuttgart: Enke, 1957, pp. 371–383.
81. KOENIG, H. *Science* 127: 1238, 1958.
82. THOMPSON, R. H. S. AND R. E. JOHNSON. *Biochem. J.* 29: 694, 1935.
83. BUEDING, E., H. WORTIS AND M. J. STERN. *J. Clin. Invest.* 21: 85, 1942.
84. JOINER, C. L., B. MCARDLE AND R. H. S. THOMPSON. *Brain* 73: 431, 1950.
85. HIROTA, Z. *Cent. Inn. Med.* 19: 385, 1898.
86. Minst. Hlth and Welf. Japan. Malnutrition and Nutrition Activities in Japan. Joint FAO/WHO Nutrition Committee for South and East Asia. 4th Meeting. Tokyo, 1956, p. 52.
87. GUERRERO, M. *Memoria*, 1904. Quoted in ALBERT, J. *Philippine J. Sci.* 45B: 301, 1931.
88. MCLAUGHLIN, A. J. AND V. L. ANDREWS. *Philippine J. Sci.* 5B: 157, 1910.
89. ANDREWS, V. L. *Philippine J. Sci.* 7B: 67, 1912.
90. CHAMBERLAIN, W. P., E. B. VEDDER AND R. R. WILLIAMS. *Philippine J. Sci.* 7A: 39, 1912.
91. ALBERT, J. *Philippine J. Sci.*, 3B: 345, 1908. Quoted in ANDREWS, V. L. *Philippine J. Sci.* 7B: 69, 1912.
92. ALBERT, J. *Acta med. Philipp.* 4: 7, 1947.
93. SALCEDO, J., E. O. CARRASCO, F. R. JOSE AND R. C. VALENZUELA. *J. Nutrition* 36: 507, 1948.
94. BRAY, G. W., *Trans. Roy. Soc. Trop. Med. Hyg.* 22: 1, 9–36, 1928.
95. HARIDAS, G. *J. Malaya Bv. Brit. M. A.* 1: 1, 26, 1937.
96. WILLIAMS, C. D. *J. Malaya Bv. Brit. M. A.* 2: 113–123, 1938.
97. CLARK, M. G. E. A. R. Municipal Hlth Off. Singapore, 89-D, 1937.
98. AYKROYD, W. R. AND B. G. KRISHNAN. *Indian J. Med. Res.* 29: 4, 1941.
99. KRISHNAN, B. G., S. RAMACHANDRAN AND K. SADHU. *Indian Med. Gaz.* 80: 10, 1945.
100. FEHILY, L. *Brit. M. J.* 2: 591, 1944.
101. TRAN VAN TU. Contribution a l'etude du beriberi infantile au Vietnam, Saigon. These: Hanoi, 1954.
102. SHARMA, D. C., *J. Trop. Pediat.* 1, 1955.
103. SATO, A. *Tohoku J. Exper. Med.* 55: 1, 1951.
104. Minst. Hlth and Welf. Japan. Malnutrition and Nutrition Activities in Japan. Joint FAO/WHO Committee for South and East Asia, 4th Meeting, Tokyo, 1956, p. 39.
105. DANN, W. J. AND W. J. DARBY. *Physiol. Rev.* 25: 326, 1945.
106. MCCOLLUM, E. V. *A History of Nutrition.* Boston: 1957.
107. BEHAR, M., F. VITERI, R. BRESSANI, G. ARROYANE, R. L. SQUIBB AND N. S. SCRIMSHAW. *Ann. New York Acad. Sci.* 69: 957, 1958.
108. HARIDAS, G. *Arch. Dis. Child.* 22: 23, 1947.
109. FEHILY, L. *Caduceus* 19: 78, 1940.
110. FEHILY, L. *Trans. Roy Soc. Trop. Med. & Hyg.*, 37: 111, 1943.
111. LOCKHART, H. S., S. B. KIRKWOOD AND R. S. HARRIS. *Am. J. Obst. & Gynec.* 46: 358, 1943.
112. KON, S. K. AND MAWSON. Med. Res. Council, Special Report, 1950.
113. ALCANTARA AND OCAMPO, Quoted from Albert and Abad.
114. ORIMO, R. *Tohoku J. Exper. Med.* 35: 35, 1939.
115. RODERUCK, C. E., H. H. WILLIAMS AND T. G. MACY. *Am. J. Dis. Child.* 70: 162, 1945.
116. RAMASASTRI, B. V. AND D. INDRAVATHI. *Ind. J. Med. Res.* 45: 447, 1957.
117. SIMPSON, I. A. AND A. Y. CHOW. *J. Trop. Paediatrics* 2: 3, 1956.
118. CHITRE, R. G. Report submitted to the World Health Organization, 1957.
119. MALCALMSON, J. G. *A Practical Essay of the History and Treatment of Beriberi.* Madras, 1835.
120. SUNDARARAJAN, A. R. *Ind. J. Med. Res.* 29: 461, 1941.
112. RODGER, F. C. *Brit. J. Ophthalmol.* 37: 11, 1953.

ENDEMIC GOITER

Introduction

SCRIMSHAW: Although the topic for discussion is endemic goiter, we won't be deprived in either the presentations or the discussions of information from experimental work on thyroid metabolism or the observations on sporadic goiter. It may be helpful if we can stipulate at the beginning, in general terms upon which we can all agree, the etiology of goiter and the general fact of its widespread distribution.

In 1952 a study group on endemic goiter convened in London (1). The WHO accepted a statement of etiology which had been published earlier in *Lancet* (2). This statement has subsequently been endorsed by the third FAO/WHO Joint Expert Committee on Nutrition (3) and by other international conferences (4). It reads as follows: "The immediate cause of simple goiter is failure of the thyroid gland to obtain a supply of iodine sufficient to maintain its normal structure and function. This failure is usually brought about by an absolute environmental deficiency of iodine. It may also be caused by factors which interfere with the availability of dietary iodine or which impose an abnormal demand on the thyroid gland." To bring this statement up to date, I would suggest adding to the last sentence the phrase, "or interfere metabolically with the utilization of iodine by the thyroid."

This concept of etiology is challenged by a few persons. It is fair to say, however, that the criticism of it has not been based on personal observations or on experimental studies, but primarily on historical records of the presence or absence of goiter in different regions at various times. While these have been interpreted as suggesting that endemic goiter is an infectious disease for which iodine is merely of therapeutic value, a critical and informed evaluation of available data does not support this conclusion.

It is also worth mentioning that wherever this concept has been tested in the field by the administration of iodine, the number and size of those goiters which were not already fibrosed has been markedly reduced. Other examples could be cited (5, 6), but I will mention briefly our Central American experience (7). Five milligrams of iodine were administered weekly to school children in El Salvador. This corresponded to the amount supplied by 10 grams of salt daily, iodized at one part in ten thousand. As figure 1 illustrates, there was a sharp drop in the prevalence of goiter within a period of 15 weeks in the experimental groups, but no change in the control groups. During the vacation period, the prevalence of goiter returned to previous levels; in the next school year treatment was resumed for a period of 20 weeks, and the frequency of goiter dropped again.

The apparent exceptions to the prompt decrease in endemic goiter with iodine treatment seem due to goitrogenic factors and will be discussed later.

To indicate the distribution of goiter, we may refer to the recent bulletin of WHO on *'Endemic Goiter'* (8). The first article in this bulletin reviews the "Prevalence and Geographical Distribution of Endemic Goiter." The first paragraph states: "Goiter, as this survey shows in detail, occurs with varying intensity in almost every country. Few countries appear to be entirely free from it. The disease has been observed in the far north, in the tropics, and in the far south. It occurs quite independently of climate, season, or weather. Moreover, in its incidence, goiter makes no distinction of race, nationality, color, creed, or class. The North American, the European, the Chinese, the Himalayan Indian, the Turkoman, and the peoples of Central and South America all suffer from it under certain conditions, some severely, some moderately, some but

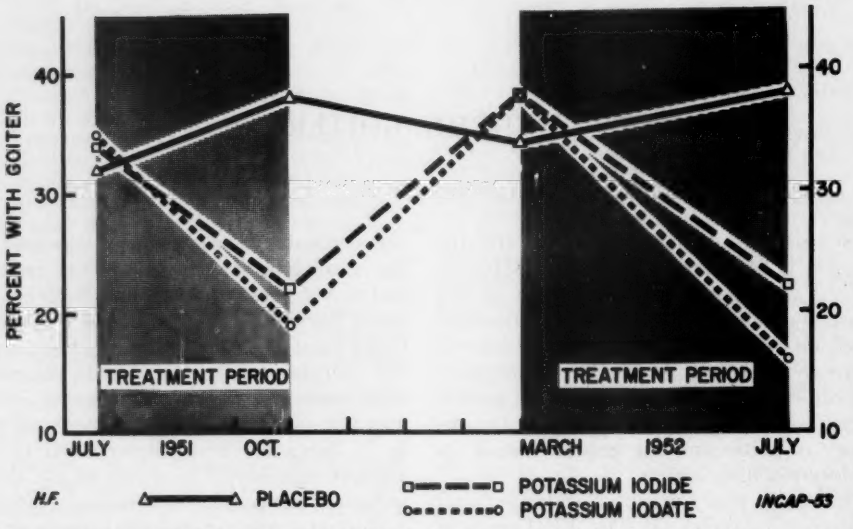


FIG. 1. Effect of suspending and renewing treatment on incidence of goiter

mildly." The article then documents this statement country by country in 145 pages and 939 references. The actual incidence of goiter in countries ranges up to nearly 40%, and without any question, there are millions of people in the world who have endemic goiter. I propose that we accept the very widespread occurrence of goiter and be concerned with the geographical distribution only to the extent that it sheds light on the mechanism of goiter.

With this widespread occurrence of goiter, one would assume that a great deal is known about its significance and its health consequences. The second article in the WHO bulletin is entitled: "Endemic Goiter: Scope of the Health Problem and Related Conditions," and written by Clements (9) who examines the nature of the evidence for the association between endemic goiter and some of the health sequelae claimed. In particular, Clements points out that the evidence for a relationship of goiter with deaf-mutism and feeble-mindedness is very uncertain. While there is no convincing published evidence, perhaps the members of this group will have unpublished data on this point. It has been stated that thyrotoxicosis and carcinoma of the thyroid are more frequent in areas with endemic goiter. The best evidence for this is Clements' own observations in Australia where he observed an increased incidence of thyrotoxicosis in goiter areas, and a drop in thyrotoxicosis paralleling a decrease in endemic goiter (10). As far as I know there is little direct evidence linking goiter with thyroid carcinoma, although Sokal has shown an association between thyrotoxicosis and subsequent carcinoma (11) of the thyroid, so by implication there must be such a relationship.

Even in the case of cretinism, which has been long cited as a classic example of the consequences of severe thyroid deficiency persisting through several generations, Clements raises the question as to whether more of the cretinism than previously suspected may not be due to familial factors accentuated by the close inbreeding in some areas in which cretinism has apparently long been associated with endemic goiter. Adverse cosmetic and mechanical effects are of course well recognized, and an increased amount of thyroid surgery is performed where people have large goiters.

In field observations in children, it has proved difficult to determine adverse consequences. The basal metabolic rate is reported to be unaltered (12, 13). Children with goiter are usually retarded in growth and development but in our experience in Central America, at least, this same degree of

retardation in growth and development can be found in children of similar dietary and socio-economic status in areas where goiter does not occur. An example is the same degree of growth retardation in children in Yucatan, Mexico, an area free of endemic goiter, as in other parts of Mexico and Guatemala. There is also the frequent statement that goiter diminishes intelligence and learning capacity; while this may be true, it still remains for someone to demonstrate it in a decisive fashion.

I. Epidemiology of Endemic Goiter

Scrimshaw: With this introduction, we may examine briefly epidemiological factors in the development of goiter. As Follis has reminded us, both host and environmental factors may be involved in any nutritional disorder and, of course, the classical epidemiological triad consists of host, agent and environment. In this case, iodine deficiency in some form is assumed to be the agent. Of the host factors, genetic influences are of major significance in sporadic cases, but presumably not of importance in determining incidence of endemic cases. Among other host factors are physiological state, including the influence of puberty, pregnancy and lactation.

Among the environmental factors, the most prominent is the difference in distribution of iodine in water and soil throughout the world. The geochemists explain the distribution of goiter on the basis of the removal of iodine from the soil by glaciation, flooding and tropical rains, with a consequent decrease in the iodine content of food and water in endemic goiter areas. There are certainly other environmental factors which exert a goitrogenic influence. The effect of calcium and hardness of water seems to be established (14). It has also been claimed in a series of reports from Germany that vitamin A deficiency increases the prevalence of goiter (15, 16). As far as I know, the original reports from Germany have not been confirmed elsewhere. In Central America, where vitamin A deficiency is common, we have tried unsuccessfully to demonstrate the effect of prolonged administration of vitamin A on the thyroid size. Finally, there are a number of known plant goitrogens and presumably many unknown ones, which can influence both the requirement for iodine and the distribution of endemic goiter. This conference should consider the manner of operation of these goitrogenic factors. Finally, nothing is known of the possible secondary influence of chronic infections and infestations on goiter incidence. I would now like to invite comments relating to the statements on etiology and distribution in order to be sure that these represent the sense of the group.

Mackenzie: The only reason for bringing up the role of compounds other than iodine at this time, is because Scrimshaw mentioned calcium, which has long been believed to be a factor that predisposed to goiter when iodine intake was low. I don't believe any epidemiological evidence could prove this to be so, because other factors associated with the same environment might well be the active causative agent. When Remington produced goiter experimentally in rats on iodide-deficient diets that are now known to contain no goitrogens and in diets in which iodide could prevent goiter, he showed that calcium had no effect on these goiters. More recently, using the same type of diet, Axelrod, Leblond and Isler (17) in Montreal produced goiters of undisputable iodine deficiency etiology in mice and completely excluded calcium as having any effect on the size of the thyroid, although they did show that the NaCl content of the diet, when the iodide intake was very low, enlarged the gland. There's one report of a few years ago by Taylor (18) that the addition of calcium to a corn type of goitrogenic diet increased the size of the goiter. However, the calcium also stimulated growth, and when the thyroid weights are calculated on the basis of body weight there is no striking difference between animals getting 2% calcium carbonate in the diet and animals getting the basal diet containing 0.36% calcium. The respective thyroid weights in milligrams per 100 grams body weight were 15.4 and 14.0.

Hazard: Reference to the hardness of water is found throughout the discussions of the geographic distribution of endemic goi-

ter. However, there is little published experimental evidence of the relation between calcium intake and goiter, excepting this paper by Taylor describing an increase in uptake of radioiodine in the thyroids of rats receiving a low iodine diet to which calcium has been added.

MACKENZIE: Taylor described differences in the uptake of radioiodide but not in the uptake of oral radioiodide. Calcium may yet be shown to play a part in the production of goiter, but two thorough investigations speak against it and I do not believe this has been demonstrated as yet.

RAMALINGASWAMI: In some recent work in which we estimated the calcium content of drinking water in areas of endemic goiter, we did not find any close coordination between the incidence of goiter and the levels of calcium. It doesn't necessarily mean that they have no relationship, but certainly there does not seem to be a straightforward relationship between hardness of water and increase in susceptibility to endemic goiter.

STANBURY: It is reassuring to have Mackenzie's explanation of Taylor's results, which seemed clear-cut, but I've never understood why calcium should or could cause hypoplasia of the thyroid. I think Taylor did have some hyperplastic changes in his gland. The role of calcium in producing goiter goes back a long way, both in epidemiological surveys and from the laboratory. There were two papers in the mid-thirties on the role of calcium in producing goiter. Thompson in 1936 (19), and Helwig in 1940 (20), both did experiments similar to Taylor's with much the same results.

MACKENZIE: Other studies were done by giving radioactive iodide and measuring the thyroid uptake. When iodide was given by mouth there was no difference between the diets with or without calcium. When iodide was given intraperitoneally, the diet containing calcium showed a somewhat higher uptake of iodide in the thyroid. Nevertheless, if we're talking about goiter, which I take to mean an enlargement of the thyroid gland, and not some functional change unassociated with differences in size, we do not have any convincing evidence that calcium in and of itself enhances the size of the thyroid, even when iodine intake is low.

STANBURY: Regarding the role of chloride, there is no question that Leblond produced remarkable changes in the thyroid in animals by loading with chloride, but this appears to be a renal phenomenon because he found that the kidney did not distinguish between chloride and iodide in resorption. Consequently, if the kidney was loaded with chloride, iodide was lost. This doesn't happen in man, however, since the human kidney appears to distinguish clearly between chloride and iodide.

SCRIMSHAW: Without reference at this time to any specific goitrogenic factors, it is worth pointing out that recognition of the occurrence of such factors has considerable practical importance. For example, the requirement for iodine given the National Research Council, U. S., range from 150 to 300 μg daily (21); by the British Medical Association 100 μg for adults and 150 for groups—specially vulnerable groups—pregnant and lactating women, adolescents and young children (22). These figures are apparently based on a limited number of iodine balance studies of normal individuals in a nongoitrogenic environment. It may be that these estimates don't apply to goitrous areas and are not adequate guides for estimating salt iodization levels. They may be no more correct that any estimate of caloric requirement from basal metabolic rate rather than from total metabolic activity.

II. Clinical Aspects of Goiter

MATOVINOVIC: In 1915 Hunziker (33) of Switzerland, and in 1954 Stanbury (55) and his colleagues published books which give the impression that goiter is a compensatory phenomenon, and we all believe it is. However, the best possible gland is a normal gland. Therefore, the enlarged thyroid in the long run may be a cause of ill health. The goitrous gland differs from normal thyroid in size and structure, and in increased tendency for deviation in function and malignant growth.

Depending on the intensity of iodine deficiency, genetic and other factors (growth,

pregnancy, lactation), the thyroid shows an increase in number of individual lobules which produce atrophy of the surrounding thyroid tissue. During this time the blood flow and the number of blood vessels in the thyroid increases. Further, arteriovenous shunts form between small arteries and large tortuous or thin-walled sinusoidal vessels in the nodules. This type of vascular change in the nodule may lead to development of ischemia or hemorrhage with degeneration, necrosis, cyst-formation and calcification of the tissue. The pathology of the nodule has been beautifully documented by N. Johnson (23, 24). The formation of hyperplastic nodules is not explained as yet, but could be due to increased supply of thyrotropin to some parts of the gland. This is based on the fact that the right lobe has a better blood supply, and therefore is usually larger than the left lobe, and contains nodules more often. The other possibility is an increase in sensitivity of some follicles to the stimulating effect of thyrotropin. Increased activity of some nodules from small amounts or no thyrotropin might lead to hyperactive, toxic nodules with hyperthyroidism. Prolonged overactivity of the hyperplastic tissue conditioned by genetic factors might be the cause for greater incidence of papillary and follicular carcinoma.

To learn about the clinical aspects of endemic goiter we can wait for a patient to come to the hospital, or we can go to his locality to see how and why his symptoms develop. The latter is more reasonable, since an individual with endemic goiter does not feel sick and does not seek medical advice. Even when the patient with goiter develops symptoms he will postpone his visit to the hospital for a considerable period because of the expense, loss of working days, etc. To see all aspects of goiter it is necessary to visit a severe endemic area in Yugoslavia where 70–75% of the inhabitants have goiter and the iodine content of the water is approximately 1.0 and 1.5 mg of 1 liter. In such areas the school children do not differ from children in nongoitrous areas, except that 80–85% of them have a diffusely enlarged thyroid gland. Girls are affected more than boys and have larger goiters than boys. The older children have larger goiters, and if classified by body weight or body frame the heavier and better grown children have the largest goiters. Probably with school children the age is a minor factor in determining the grade of enlargement of the thyroid. It is found that goitrous children have poorer grades than normal children.

Among adolescents from 15–20 years, girls again lead in the frequency and degree of the enlargement of the thyroid, but neither boys nor girls have symptoms. However, even at this age in some families nodules can be found in the thyroid. After age 20 the natural course of goiter in man is more stationary, with slow growth of the gland. Nodules develop between the ages of 35–45; there are few symptoms and these are not severe. However, in the female, frequent pregnancies followed by lactation cause repeated over-activation and increased rate of growth of the thyroid. Such women often have large multinodular goiters between the ages of 25–35 years. From this time on, the large (5–10 times normal size) goiter may compress the trachea and larynx, producing shortness of breath and cough, the recurrent nerve producing hoarseness (vox strumica), cervical nerves with occipital neuralgia, brachial plexus with right shoulder syndrome, paresthesia and weakness of the right hand, sympathetic nerves with Horner's syndrome, and, rarely, the vagus nerve with attacks of bradycardia and syncope. The goiter often causes engorgement of the neck veins with congestion of the face and increased intracranial pressure with discomfort during heavy work. The increased arterial flow with arteriovenous shunt may lead to hemorrhages, producing a sudden increase in size of the nodule with severe shortness of breath due to compression of the trachea.

The prolonged increases in blood flow with arteriovenous shunt in very large goiters can be a factor in the development of congestive failure in the elderly patient with arteriosclerotic or valvular heart disease (Kropfherz). The upper pole of an enlarged left lobe can compress the esophagus and produce dysphagia.

Compression symptoms may become more marked as time passes. As the growing gland compresses the sternocleidomastoid muscles (especially during heavy work) it is forced into the thorax because of its weight

and by negative pressure during inspiration until it lies beneath the sternum. Then, during heavy work, especially if the hands are raised above the head, the patient develops great difficulties in breathing. However, compression symptoms may in time be alleviated because the goiter, or better, its right lobe, remains permanently in the thoracic location. Such patients will notice a decrease in size of the goiter and may be awakened during the night with an attack of dyspnea produced by pressure of the intrathoracic goiter on the trachea. Therefore, the diagnosis of a large substernal lobe of the thyroid should be considered in any patient admitted to the hospital for nocturnal dyspnea in whom only a large left lobe of the thyroid is palpable.

All these compression symptoms usually progress with the age of the patient and ultimately 3–5% of all patients with goiter ask for medical and finally surgical help. On the other hand, the goitrous patient from an endemic goiter area seeks treatment for cosmetic reasons relatively infrequently.

About 20% of multinodular goiters become toxic. The symptoms consist of loss of weight, palpitation of the heart, absolute arrythmia and/or congestive failure. Forme fruste with chronic diarrhea or anxiety states up to psychosis are not infrequent. Contrary to hyperthroidism, exhaustion of the thyroid with hypothyroidism is quite rare and is observed only in old women.

Anaplastic carcinoma seems to be as common as in non-goitrous thyroids. Papillary and follicular carcinoma are more frequent in endemic goiter areas. This was pointed out by Wegelin. Carcinoma has to be suspected if a patient, after the age of 30, develops recurrent goiter in 1–2 years instead of 5–10 years. Otherwise, a patient may have no symptoms for a long time.

In addition to these numerous complications of endemic goiter, there are the complications of surgical therapy of the goiter: paralysis of the recurrent nerve in 1%, hypoparathyroid tetany in 0.5%, hypothyroidism in about 1%. Recurrent goiter occurs in 5.9% and mortality in 0.2%.

Finally, in our country endemic cretinism and deaf-mutism are very common. Despite the experience that two events that occur at the same time or place do not have to be in cause and effect relationship, one cannot forget that both of them are seen together in many families with endemic goiter.

Having all these symptoms and complications in mind, one can imagine how much unhappiness and suffering is caused by the so-called simple goiter. It is not surprising that 100 years ago, before surgical therapy and iodine prevention of goiter, one author thought the following sign should be erected on the roads to endemic goiter areas: "Lasciate speranza voi ch'entrate"—(Abandon all hope, ye who enter here).

RAMALINGASWAMI: It is fairly well recognized that in the evolution of endemic goiter nodules develop with a certain amount of frequency. I should like to ask Matovinovic for any explanation for the occurrence of nodules, because we know that the initial reaction of the thyroid gland is a diffuse hyperplasia, and it is only after some time that certain areas within the gland are converted into nodules. I wonder if the studies on the circulation through the gland provide any clue; is it possible that areas which have an intact circulation, under the influence of the thyrotropic hormone, develop nodular hyperplasia in the course of time?

It is a well accepted finding that some of the nodules tend to develop toxic features. Apparently the incidence of toxicity and of nodular goiters in different parts of the world is variable. For example, in the Himalayan area we expected to find a lot of thyrotoxic manifestations because of the existence of nodular goiter, but we were not able to find many cases, although they do occur. I wonder whether the liability of the nodules to develop toxic features is related in some way to the etiological factors, that is, certain positive factors in the diet producing or having a greater tendency to lead to toxic manifestations.

SCRIMSHAW: Before referring this question to Matovinovic, I'd like to point out that in the Latin American surveys there is a great variation in the occurrence of nodular goiter in children. In some villages more than half of the children may have goiters with half of these containing nodules. There are even occasional villages where thyroid nodules can be found among the preschool children. In other villages where the

total amount of goiter appears to be the same, there may be no nodules clinically detectable. Because the occurrence of nodules does not bear a consistent relationship to thyroid size or goiter prevalence in the population examined, Kimball's suggestion that nodules arise as a result of a severe maternal iodine deficiency (25) is an attractive hypothesis.

MATOVINOVIC: I have no experimental knowledge regarding the development of toxic nodules. The only thing that we can say is that a toxic nodule is more common in the female than in the male and that in an endemic goiter area toxic nodules occur at an earlier age in the female than in the male. There are some indications that blood supply might play a role and with it a greater stimulation with TSH, for toxic nodules are more common in the right lobe where more blood is given to the gland. The role of vessels in development of nodules was shown by Johnson (23, 24).

For the incidence of nodules in children we have no explanation. In collaboration with Prebig (56), we examined 20,000 children in the primary schools of Zagreb, Yugoslavia. The children were of the same age. The sex incidence was the same, approximately 9,500 for each sex. Boys had goiters in 46%, girls in 51%. We have found that in older children with greater body weight the incidence of goiter was more common. In that group there were nodules in less than 1% of children. However, Zagreb is not considered an endemic area.

TEJADA: I think that there are some indications (26) that in the center of mountainous goiter regions the incidence of hyperthyroidism is less than in level regions such as Northern Europe or North America. This confirms Ramalingaswami's statement that in the Himalayas the incidence of thyrotoxicosis is not great. As far as Central America is concerned, the incidence of thyrotoxicosis is low.

STANBURY: When considering what causes *toxic* nodular goiter, all one can do is raise questions. I doubt if we are going to be able to provide any answers in the immediately foreseeable future. I should like to describe a disease which we have in Boston. It occurs first in women who, during adolescence, develop diffuse hypoplasia of their thyroid glands. This becomes a fixed disease in their 20's and 30's and by their 30's and 40's they are developing nodular goiter. When they are 50 or 60 or 70 they have developed toxic nodular goiter. There are a great many people who go through this lifelong cycle, and I think this disease probably exists in 2% of the population in eastern Massachusetts. I raise the question as to just what is 'endemic' goiter and how is it defined. Certainly, at 2% incidence level the population in Boston is only quantitatively different from places where there is a 50% incidence. I don't know where one draws the line to delimit endemic goiter.

SCRIMSHAW: No one seems willing to try to answer Stanbury's question. Perhaps we should expand our definition of endemic goiter to include such cases, but I'm afraid that it would cause confusion.

III. PATHOLOGY

A. *The Pathologic Anatomy of Endemic Goiter Before the Introduction of Iodized Salt*

UEHLINGER: The normal thyroid gland consists of lobules separated by fibrous connective tissue. The lobules consist of spherical follicles filled with acidophilic colloid. The follicles are surrounded by reticular connective tissue, blood and lymphatic vessels and nerves. In older age the central areas of the colloid may show various grades of basophilia, probably due to nucleoproteins. Basophilic colloid contains less iodine than acidophilic colloid. The follicles are lined by cuboidal or flattened epithelium. The central follicles show some cylindrical epithelium. There is no basement membrane between epithelium and supporting connective tissue.

Thyroid growth goes on during childhood and stops with puberty. The weight of the normal thyroid is still controversial. Kloeppel (27) gives the following data from goiter-free areas: newborn, 5 gm; adults between 21 and 50 years, 34–41 gm; adults between 51 and 65 years, 30–32 gm.

Goiter is defined as an enlargement of the thyroid to compensate for secretional insufficiency and decreased activity to store iodine. The enlargement may be due to a diffuse or a focal proliferation of the glandular tissue with formation of nodules. The main differences between diffuse and nodular goiters are as follows: in *diffuse* goiter the normal structure is well preserved. The relationship between follicles and blood vessels is the same as in normal thyroid. In *nodular* goiter there is a progressive isolation of the nodular tissue with progressive growth of the nodules. The nodules are surrounded and separated from the normal lobular tissue by a distinct capsule of connective tissue. The growth proceeds at the periphery while the central area undergoes degeneration. The central follicles gradually disappear and are replaced by connective tissue. The central scar may undergo further changes as hyaline degeneration, calcification, ossification and bleeding. Finally, the whole nodule may be transformed into a thick-walled cyst lined by a few layers of small follicles.

Aschoff (28) does not think the nodules are related to the goiter problem itself. He believes the nodules are benign tumors, documenting their independency by spontaneous structural changes and lack of senile atrophy. Contrary to the explanation of Aschoff, Wegelin (29, 30, 31) believes that the formation of nodules and the nodular goiter are intimately connected with the goiter problem. The same agent that stimulates the diffuse hyperplasia stimulates the growth of nodules. McGavack (32) expressed the same conclusion in 1955: "Thyroid nodules arise as a result of excessive stimulation of, or load upon the thyroid glands."

TABLE 1. CLASSIFICATION OF ENDEMIC GOITER (WEGELIN)

A. STRUMA DIFFUSA
   1. Struma diffusa parenchymatosa (microfollicularis)
   2. Struma diffusa colloides
B. STRUMA NODOSA HYPERPLASTICA (nodular hyperplasia)
C. STRUMA NODOSA ADENOMATOSA
   1. Struma nodosa parenchymatosa (trabecularis, tubularis, microfollicularis)
   2. Struma nodosa colloides macrofollicularis
D. STRUMA DIFFUSA ET NODOSA (mixed forms)

TABLE 2. CLASSIFICATION OF ENDEMIC GOITER (ASCHOFF)

A. *Hyperplasia of the thyroid gland* (Struma diffusa)
   1. Struma diffusa parenchymatosa neonatorum
   2. Struma diffusa colloides macro-follicularis
      (a) Proliferans. Hyperthyroidism (slight and severe degrees). Thyroidism from therapeusis. Formes frustes. Basedowoid. Thyroidic constitution
      (b) Non-proliferans. More indifferent, suppression, slight hypothyroidism to cretinism
   3. Struma diffusa colloides microfollicularis (normal mountain thyroid of Wegelin)
   4. Struma diffusa parenchymatosa Basedowians. Hyperthyroidism of severe degree
      (a) With Basedow triad
      (b) Without Basedow triad
   5. Struma diffusa colloides Basedowificata
B. *Hyperplastic adenomatous form*
   6. Struma diffusa et nodosa colloides hyper- and hypothyroidism (proliferans or non-proliferans)
C. *Adenomatous form*
   7. Struma nodosa simplex. Hypothyroidism
   8. Struma nodosa Basedowifacata. Hyperthyroidism

The intermediate phase between diffuse and nodular goiter is called *nodular hyperplasia*. Characteristic of this type is the multiplicity of proliferating centers without separation by capsules. Wegelin gives in his most recent contribution to the goiter problem the following classification (table 1). This classification avoids too much differentiation. I have used this classification for several years with much satisfaction. In Germany the classification of Aschoff is still used (Table 2). Aschoff gives a more detailed differentiation and with a clear-cut separation of the diffuse and adenomatous types.

There is general agreement that endemic goiter begins with enlargement of the thyroid of the fetus. In iodine-deficient regions the goiter of the newborn corresponds to the diffuse microfollicular epithelial hyperplasia. The lobular pattern is well preserved. The number of small follicles is increased. The follicles are colloid free. The interstitial tissue is markedly congested. The weight of the endemic goiter of the newborn shows a great variability in relation to the different

congestion. In any case, a weight of 6 gm is abnormally high.

During childhood the number of follicles increases. During puberty there is a marked enlargement of the thyroid due to the storage of eosinophilic colloid, and often combined with nodular hyperplasia. The weight of the goiter in puberty is about 50 and 150 gm.

In adults the nodules increase with age, therefore, in adults there is a great variability in weight and types of goiter. The weight and the number of nodules reflects the endemicity of goiter. The principal types of goiter after puberty are the diffuse stationary colloid goiter, the diffuse colloid goiter with nodular hyperplasia and the nodular goiter.

In old age the weight of the thyroid decreases. This decrease is related to the senile and pressure atrophy of the lobular tissue. At the same time the nodules may increase in size by proliferation and hemorrhages.

Aschoff divides the whole life cycle of the thyroid in seven periods, as follows:

1) Thyroid enlargement of the newborn
2) Regression during infancy
3) Thyroid enlargement during pre-puberty and puberty
4) Regression in post-puberty
5) Normal gland of the adult during the height of life
6) Age atrophy of the gland in the decline of life
7) Possible rejuvenation of the thyroid function in old age.

The historical review of goiter investigation deals with four problems:

1) The behavior of the thyroid gland in coastal and in mountain areas
2) The relationship between goiter and cretinism
3) The relationship between goiter and 'strumitis'
4) The relationship between goiter and thyroid cancer.

The behavior of the thyroid gland in different regions was studied at the beginning of the 20th century by Aschoff and his pupils. The results were as follows: The mean weight of the thyroid in coastal regions is markedly lower than in mountain regions. In coastal regions there are no significant changes in the weight of the thyroid during its life cycle; however, we can recognize the same seven periods that are found in the mountain goiter regions.

The relationship between goiter and cretinism was studied by Wegelin and reported at the Second International Congress of Goiter in 1938. Wegelin came to the following conclusions:

1) Endemic cretinism is only to be found in districts where goiter is endemic, in a geographically narrower range than goiter alone and only where endemicity is high.

2) The thyroid gland of the cretin almost always contains adenomatous nodules which vary greatly in size. They are almost without exception microfollicular and contain little or no colloid.

3) The behavior of the original lobular tissue between the nodules is always the same. The epithelial cells of the gland undergo a more or less severe degenerative change regarding nucleus and cytoplasm, even before birth.

4) The thyroid gland of a cretin is, as regards function, an inferior organ, although a part of its function may be taken over by adenomas.

5) In the etiology of endemic cretinism external injuries of the thyroid gland must be taken into account as well as inbreeding.

In goiter areas 'strumitis' is by no means rare. The most common types are the granulomatous forms (giant-cell strumitis). Not so common is the sclerosing strumitis (Riedel). In goiter regions struma lymphomatosa (Hashimoto) is extremely rare. Tuberculous, syphilitic and mycotic strumitis are even more rare.

The relationship between goiter and thyroid cancer has been studied in Austria and Switzerland. Thyroid cancer is much more common in goiter regions than in goiter-free regions. Wegelin makes the following statement on thyroid cancer in the autopsy cases:

Bern: 1:96 = 1.04%
Wien: 1:363 = 0.27%
Berlin: 1:1033 = 0.09%
USA: 1:928 = 0.107%

Thyroid cancer in Bern is ten-fold more frequent than in Berlin. The relationship between goiter and carcinoma of the thyroid is confirmed by the histologic structure of some types of thyroid cancer, especially the

'metastasizing adenoma' and the hemangioendothelioma. Both tumors always develop in nodules.

Our 'metastasizing adenoma', called well-differentiated adenocarcinoma in the United States, is histologically characterized by subcapsular epithelial proliferation with progressive differentiation in follicles towards the central areas. This tumor can secrete hormone, which could be demonstrated in the famous case of Eiselberg. I personally had the opportunity to observe the hormonal activity of a metastasizing adenocarcinoma.

The hemangioendothelioma is only known in goiter regions. The tumor develops from peculiar thick-walled vessels in the central scar. Histologically we find a diffuse or more trabecular proliferation of spindle cells with secondary space formation. The final result is a mixture of sarcoma-like structures with blood-filled spaces lined by tumor cells. Hemangioendothelioma is an extremely rapidly growing and metastasizing tumor.

I wish to add some actual goiter statistics reflecting my personal experience in Zurich. In our autopsy material 1946–1951 with a total of 5,408 autopsies, we found 929 normal thyroids. The number of thyroid glands showing diffuse hyperplasia was 1, 421; of nodular goiter 3,037; of thyroid cancer 21. These figures are impressive and demonstrate that the goiter problem is still a very interesting one which requires further investigation and information.

B. *The Influence of Iodine on Swiss Goiter*

UEHLINGER: Lack of iodine is the main but not the exclusive factor, in the etiology of goiter. To compensate for the lack of iodine the cantonal Swiss governments began, step by step, the introduction of iodinized salt so that each 1000 gm of salt contains an additional 5 mg potassium iodide. The remarkable results are as follows:

The frequency of goiter in the newborn with a weight more than 3 gm diminished in Bern from 88% in 1924 to 42% in 1936, the frequency of the neonatal goiter with a weight of 6 gm or more diminished from 42% in 1924 to 5% in 1936.

Between 1946 and 1951 the Swiss Health Service made, with the assistance of the Departments of Pathology of the University Hospitals in Basel, Bern, Lausanne, Geneva and Zurich, a comparative study of the thyroid, concerning the weight and morphology. Comparing with earlier investigations at the beginning of the 20th century, the statistical evaluation of the information given by the different pathology departments shows the following facts:

1) Marked reduction of the mean weight of the thyroid
2) Diffuse and nodular goiter still occurs but at a later age
3) Goiter weighing 500 gm and more (giant goiter) has disappeared
4) Endemic cretinism is a disappearing disease
5) The goiter of the newborn (diffuse epithelial hyperplasia) is still decreasing and shows further diminution to 10–15%.

The results vary in different regions. In the French part of Switzerland the results are less striking than in the German part of Switzerland.

The development of goiter still follows the pattern outlined by Aschoff and Wegelin. The first phase of diffuse epithelial hyperplasia is followed by a second phase of colloid storage and nodular hyperplasia between the age of 20 and 40. Nodular goiter is a late manifestation of endemic goiter and replaces, with increasing age, the diffuse and nodular hyperplasia with colloid storage.

There is no change in the frequency of thyroid inflammation (granulomatous and sclerotic thyroiditis).

There is no clear-cut diminution in thyroid cancer. The time elapsed between the introduction of iodinized salt and cancer age is still too short to influence the rate of thyroid cancer.

Iodine does not influence the growth of the nodules (adenomas).

Iodine intoxication occurs, but is rare.

C. *Relation of Thyroid Changes to Nutritional Status*

TEJADA: The evidence is quite consistent that chronic malnutrition produces in humans serious endocrinological disorders, characterized clinically by lowered functional activity (33, 34). Histopathological changes in the thyroid have been reported in adults and children suffering from malnutrition and there is some evidence that

thyroid activity is lowered in patients with malnutrition.

Oberndorfer (35), Lamy *et al.* (36), Zubiran and Gomez Mont (143) and Paullada (34) found changes in adult thyroids consistent with atrophy. Uehlinger (37) and Stein and Fenigstein (57) found relatively few changes in the thyroid. In children with malnutrition, Lucien (38), Marfan (39), Thompson (40), Nicolaeff (41) and Davies (42) found changes which they interpreted as evidence of thyroid atrophy. On the other hand, in children under 5 years of age, whether malnourished or not, Ambrosius (43) was not able to detect differences; however, in malnourished children over 5 years of age, he reported the presence of flat epithelium and large follicles full of colloid.

In the majority of these studies, the changes in the thyroid have been considered in relation to malnutrition, disregarding other factors which could alter the structure of the gland. Other studies have been concerned with the end stages of thyroid disease, disregarding histological changes observed in glands without gross pathology. Since the prevalence of both malnutrition and goiter as well as the presence or absence of goitrogenic factors varies from area to area, many of the previous studies have been contradictory and inconclusive.

I am presenting results from a pathological study of the thyroid changes observed in Guatemala, in an attempt to explore the possible relation between the nutritional status and the thyroid lesions found at necropsy.

During 1957 a total of 357 thyroid glands were obtained at necropsy from the General Hospital in Guatemala City. The patients admitted to this hospital came from all departments of the country, the majority are of Mayan extraction and all belong in the low economic group.

Histologically, the thyroids can be arbitrarily classified in four groups. In many cases, however, there is transition from one group to another and this results in some difficulty in classification. The histological groups considered in this study are as follows:

a) Normal Thyroid. The follicles are large, measuring up to 500 microns in diameter. The colloid material is abundant and stains deeply. The cells lining the follicles are commonly low cuboidal and there is no interstitial fibrosis. In children under 5 years of age, there is usually some fibrosis present, as it is also the case in adults over 50 years of age because of the normal physiological involution of the gland. The majority of the so-called 'normal' thyroid glands in Guatemala have acini of small size, scanty and thick colloid, poor blood supply and increased fibrous stroma. The general appearance of the thyroids in Guatemala is, then, quite similar to the so-called senile or physiological involution of the thyroid. The same characteristics have been described in the 'normal' thyroid glands in Switzerland (44).

b) Hyperplasia of Thyroid. The stores of colloid are exhausted and the follicles are much smaller in size but the lumen is always present. The low cuboidal cells lining the follicles become columnar, increasing in size and number, but the stroma is not fibrotic.

c) Thyroid Atrophy or Thyroid Exhaustion. The stores of colloid are exhausted and the follicles are much smaller in size. The lumen is present or absent and the follicular cells form small clumps without any evidence of central lumen. The cells lining the follicles are flat and atrophic. The stroma usually is quite fibrotic, but there is no evidence of inflammatory reaction.

d) Thyroid Desquamation. The colloid is absent; the follicles are usually a good size but quite difficult to recognize without the aid of either collagen or reticulum fiber stains since in this case there is marked intraluminal desquamation of the cells lining the follicles. The thyroid desquamation as described probably occurs post-mortem, but may be conditioned through predisposing changes in the structure of the thyroid which occur before death. In some cases desquamation is evident a few hours after death, but in many others is not apparent even 15–20 hours post-mortem.

This classification of the thyroid parenchyma is completely arbitrary and used only to facilitate the discussion. There are so many exceptions that it is impossible to determine the functional state of the gland in all cases by histological examination alone.

All the thyroids with no gross pathology such as goiter or nodules were compared

histologically and arbitrarily classified into the previously described four groups: *a*) normal thyroid, *b*) hyperplasia, *c*) atrophy or exhaustion, *d*) desquamation. Since malnutrition differs in children and adults, the two groups were separated for analysis.

Five thyroids from children less than 10 years of age and 27 from adults over 20 years of age served as controls. These thyroids were obtained from the Charity Hospital and the Medicolegal Department of New Orleans and all were from well-nourished persons who died without emaciation or chronic illness. New Orleans represents a seacoast location in a non-endemic goiter area of the U.S.A.

a) *Thyroid changes in adults.* Our data indicate that the distribution of the histological thyroid types considered in this study differs significantly in New Orleans and in Guatemala. They also indicate that there is no difference in the distribution of histological types in regard to nutritional status in Guatemala. This may be indicative of the fact that the observed differences are not due to nutritional status per se. The differences observed between Guatemala and New Orleans might be explained as a result of iodine deficiency, nutritional status or a combination of the two conditions. Unfortunately, we lack, at present, information on a well-nourished population from an endemic goiter area to further speculate on possible causes of these changes.

b) *Thyroid changes in children with kwashiorkor and marasmus.* Among the 80 children less than 10 years of age which were severely malnourished—18 had kwashiorkor and 8 had marasmus. These two conditions have been well described by many authors (42, 45, 46).

Thompson (40), Lucien (38), Marfan (39), Nicolaeff (41) have described gross and histological changes in the thyroid in children with malnutrition. In the present study the thyroids in cases of kwashiorkor and marasmus were found to be histologically similar, and it was impossible to differentiate changes between the two conditions. Among 18 cases of kwashiorkor, there were 8 thyroids with atrophy and exhaustion, 5 with hyperplasia, and 5 were normal. In the 8 cases of marasmus, there were 5 glands with atrophy and exhaustion, 1 with hyperplasia, and 2 normal. The remaining thyroids from the other children from Guatemala did not show any difference from the ones with kwashiorkor and marasmus. Five well-nourished children from New Orleans, which served as controls, were found to have normal thyroids.

In normal children there is some fibrosis of the interstitial tissue and the diameter of the follicles is smaller than in adults (47). In order to study possible differences in the distribution of follicular size, the diameter of 400 follicles was measured in each thyroid from the cases of kwashiorkor, marasmus and the New Orleans children.

In cases of kwashiorkor and marasmus the majority of the follicles measured less than 60 microns and only few follicles measured more than 90 microns. In the New Orleans children the size span of follicles is wider than in the cases of kwashiorkor and marasmus, and follicles which measure more than 90 microns are frequent. Hellwig (47) has measured the average diameter of the follicles in thyroids from Kansas, and found that at the time of birth the acini were solid or had a very narrow lumen without colloid. The size increased rapidly during the first year of life, reaching 160 μ as early as the fifth year. In our series the average diameter of the follicles was 42.6 and 36.2 microns for children with kwashiorkor and marasmus respectively.

Guatemala (where both goiter and malnutrition are prevalent) offers a good opportunity to determine if there is any difference in the thyroid changes which may be attributed to malnutrition or goitrogenic factors and deficiencies. The present study has merely explored the preliminary aspects of the problem and partial further studies are underway. From the partial report, however, the following conclusions can be tentatively drawn:

1) There is a real difference in the histological structure of the Guatemalan and New Orleans thyroids. This difference may be due to different nutritional backgrounds, different prevalence of goiter in the two populations, or a combination of both factors.

2) When thyroids from Guatemala alone are compared, no definite statistical differences can be attributed to nutritional

causes. There is, however, a slight tendency for more nodules to occur in undernourished adults than in well-nourished adults, while frank goiter seems to be more frequent in the latter group. No histological difference was observed.

3) Further geographic pathological studies are necessary to establish conclusively if there is any relation in the pathology of the thyroid gland and the general status of chronic malnutrition.

HAZARD: Morphologic studies made by Kaufman and myself in the Cleveland area (48) showed that the incidence of nodules in the thyroid gland was 49.9% for women and 38.4% for men at autopsy. Insofar as the non-nodular glands were concerned, 8.1% weighed more than the 35 grams, which we accepted as the dividing line between the thyroid that was non-goitrous and the goitrous gland. The nodular changes varied from those occurring as a solitary nodule, which appeared in about one-fourth of the total nodular group, to those that had many nodules. Of the glands that were non-nodular, as I stated, about 8% were enlarged, most of the others were normal in appearance. A few did reveal some fibrosis, similar to that just shown by Tejada. Rare glands showed a microfollicular structure rather than follicles of usual size. These, however, were so exceptional that I do not believe they need comment. I would be interested in knowing from Tejada the incidence of nodules in the glands that he examined.

TEJADA: A total of 359 thyroid glands were examined. Eighty glands which were from children in the first decade of life, all of which showed no signs of goiter or nodules, were not included in the final calculations. The other 279 thyroid glands were from persons over 10 years of age. Of these, 128 glands were normal on gross examination, 62 were diffuse goiters, while 34 thyroids weighed less than 35 grams but contained one or more nodules. From these data it is evident that 89 glands, or 31.8%, contained nodules.

The relative frequency of thyroid glands with nodules appears to be higher in the series of Rice (49) and Coffey *et al.* (50), probably because surgical material was considered and the glands were clinically abnormal. The work of Hull (44) shows a large per cent of nodules in necropsy material; his data, however, are not comparable with ours, since in the present study small nodules which were not well defined on gross examination and which measured less than 5 millimeters were not considered. The work of Schlesinger, Gargill and Saxe (51), on the other hand, is quite comparable with our data. The differences observed in these two series may be a reflection of the non-endemic goiter situation in Massachusetts against the endemicity of goiter in Guatemala. The data of Hellwig (47) and Nolan (52) also are quite comparable with ours.

On listing the relative frequency of goiter by decades, it is apparent that as age advances there is a transition from normal-looking glands to diffuse goiter and later to nodular glands. This has been observed previously by Wegelin (53) in Switzerland. It appears that in the 2nd, 3rd, and 4th decades the numbers of thyroid with diffuse goiter or with nodules increase steadily, while after the fourth decade there is a decrease in the number of diffuse goiters with a corresponding increase in the number of nodular glands.

There is no significant evidence that the distribution of thyroid conditions varies according to the present classification of the nutritional status. There are, however, slight indications that changes in the thyroid because of differences in nutritional status may still be possible, since a larger number of nodules were found in the thyroids of malnourished adults, while frank goiters seemed to occur more frequently in the non-malnourished subjects.

HIGGINSON: I would like to ask what we mean by a normal thyroid gland. Certain slides shown by Uehlinger as representing a normal gland I would regard as abnormal in my South African experience. I have always regarded flattened epithelium in a thyroid as representing some degree of glandular dysfunction. Secondly, in kwashiorkor, we found some degree of atrophy of thyroid in association with atrophy of other exocrine and endocrine glands. Thyroids from older children and teenagers showed usually no excessive atrophy, fibrosis or nodularity, but numbers were small. My

general impression is that a nodular thyroid gland is not unduly frequent in the Bantu in Johannesburg, although there is definitely endemic goiter in parts of the Transvaal. We also have accurate statistics for thyroid cancer in the Bantu of Johannesburg and it is significantly less than in America. Lastly, it is widely stated by physicians that thyrotoxicosis in the Bantu, whether primary or secondary, is exceedingly rare. Accordingly, in the Johannesburg Bantu, whose diet is believed inadequate by European standards, there is no striking increased frequency of goiter or its complications.

RAMALINGASWAMI: I was interested in the observations made by Tejada on the structure of the thyroid gland in kwashiorkor and marasmus. We have kept a group of monkeys on low protein diet for a fairly long time. After 4 months of depletion, we observed that the thyroid glands were either normal or there was only a slight degree of atrophy. We have never seen hyperplasia in these glands. I thought this might be of interest, because here one is dealing with a pure protein deficiency. We were stimulated by the work of Stanbury to look more closely into the thyroid glands in protein deficiency in the light of his observations on the enzymatic reactions involved in the synthesis of the thyroid hormone.

Uehlinger made a very important point about being able to relate the histological pattern of metastatic thyroid carcinoma to a preexisting endemic goiter. He mentioned that the degree of differentiation of the carcinoma was a point in favor of regarding the cancers as possibly related to endemic goiter. I'd like to know how the incidence of the fully differentiated carcinoma of the thyroid in this series compares with studies from non-endemic areas in Switzerland and elsewhere. He also mentioned that there were 21 cases of carcinoma of the thyroid in about 5,000 autopsies. Again, I'd like to know how this compares with the data from non-endemic areas in Switzerland, and elsewhere.

SCRIMSHAW: The possibility which Ramalingaswami mentions regarding thyroid changes observed in kwashiorkor, was the motivation for the pathological studies which Tejada has described. It was suspected that many of the changes attributed to protein malnutrition were really associated with iodine deficiency in the general population. There was the precedent that some of the liver changes in kwashiorkor in Africa attributed to protein deficiency have subsequently been shown not to occur in kwashiorkor in other areas. It is certainly the general implication of Tejada's work that many of the thyroid changes are not in fact specific to kwashiorkor, but are also present in the general population.

UEHLINGER: The question of Ramalingaswami concerns the different types of cancer of the thyroid. I have statistic data on struma maligna from St. Gall. In 10 years we found 107 carcinomas of the thyroid. Thirteen of these were hemangioendotheliomas and 38 well-differentiated adenocarcinomas. Both tumors only develop in pre-existing nodules, so both must be, in some way, related to nodular formation in endemic goiter. Autopsy material doesn't reflect the real incidence of thyroid carcinoma because these patients often leave the hospital and do not have an autopsy. Biopsy material is more representative of the true incidence of thyroid cancer. I am sorry I cannot answer the question about the frequency of thyroid cancer in goiter areas and in goiter-free areas in Switzerland.

HIGGINSON: Tejada, does the term 'malnourished,' as applied to autopsy material, refer to individuals dying from a disease due primarily to nutritional deficiency, or does it refer to persons dying from a wide variety of debilitating diseases such as cancer, chronic tuberculosis, etc.? Such patients may be malnourished terminally, but such a debilitating illness does not necessarily apply to a long period of poor nutrition.

TEJADA: I'm sorry I didn't have time to discuss how we classified our cases from the nutritional point of view. This was determined in each case on the basis of weight, height, thickness of subcutaneous fat, deposits of fat in the mesentery, mediastinum and retroperitoneum, as well as from the color and appearance of the muscle tissue, and by the past dietary history as recorded in the clinical chart. The majority of the adult low income population of Guatemala is definitely undernourished. And in our material the majority of our undernourished

cases are from patients with tuberculosis, infectious diarrhea, parasitic diseases and respiratory infections.

DARBY: I also object slightly to this use of the term malnourished. It seems to me from this description which Tejada has given us that he's talking primarily about caloric malnutrition or caloric status rather than nutritional status.

UEHLINGER: May I add some facts about my experience of the influence of chronic caloric malnutrition in prisoners from German concentration camps. I didn't observe any significant change in the thyroid in those patients. At the end of the first World War Oberndorffer (35) described varying degrees of atrophy of the thyroid. In my case material I couldn't confirm this statement.

ANDRUS: I would like to ask Uehlinger if he feels that his incidence of hemangioendothelioma is higher in proportion to his thyroid malignancy cases in general than elsewhere in the world, and also, if in his nongoitrous thyroids he sees any of the baseline changes that Tejada has shown, i.e., the variation in size of follicles and the increased amount of fibrosis.

UEHLINGER: The hemangioendothelioma is a 'Swiss type' of thyroid cancer. I looked in the files of the Armed Forces Institute of Pathology in Washington, and there was no specimen of thyroid hemangioendothelioma. Later I sent some typical specimens to the Armed Forces Institute, and most of the consultants called this tumor a vascularized sarcoma. In my belief, the hemangioendothelioma of the thyroid is quite a distinct tumor, always related to nodular formation and has to be separated from the common types of sarcoma. I have not observed in Switzerland the diffuse fibrosis and diffuse atrophy showed by Tejada.

DARBY: Uehlinger, is there evidence concerning the influence or changes in incidence of cretinism since the introduction of iodinization? This is a very important point to establish from the standpoint of preventive medicine, because if iodinization has been associated with a great reduction or disappearance of cretinism, regardless of any genetic factors involved in the production of cretinism, then this is one of the strongest possible arguments for iodinization.

UEHLINGER: The statement holds for the disappearance of endemic cretinism only and not for sporadic cretinism. Sporadic cretinism still occurs in Switzerland.

SCRIMSHAW: I think that this disappearance of cretinism in Switzerland would merit any further comments that people may have to make; we should know that Wegelin also recently reported from Switzerland that no more cretins were being born.

HAZARD: I would like to briefly discuss the anatomical pathology of the thyroid in sporadic cretinism and what it might possibly be in endemic cretinism. I haven't seen endemic cretins myself, so I have to ask Uehlinger or Tejada if the sporadic cretin has a gland which is much different from that of the endemic cretin. The thyroids of sporadic cretins are hyperplastic and become nodular early. The incidence of nodules is very high; the nodules themselves are hyperplastic with small, closely-packed follicles. I wonder whether, since we're speaking about cretinism disappearing in the Swiss area, the endemic cretin has a gland that looked like the sporadic gland that I have just described.

STANBURY: For some time I've been carrying on vigorous correspondence with Professor Aurilio Costa of Turin, concerning the nature of endemic cretinism. He has the advantage of me because I have had no experience in the field. Professor Costa seems to think that in addition, or perhaps quite apart from iodine deficiency, there is another or other factors operating in the production of the cretin. He thinks so primarily because he observes that the protein-bound iodine concentration in the serum may be normal in established endemic cretinism, and because these subjects may show a normal or enhanced affinity for radioactive iodine. It seems to me that these observations are not necessarily relevant, because I suppose that the damage was done permanently and irrevocably during fetal or early neonatal life when the gland was producing no hormone. I should like to ask if there is the slightest evidence that endemic cretinism—apart from the occasional athyroidic cretin who can arise in any community, and apart from familial cretinism of the type which Hazard was referring to—is due to any cause other than iodine deficiency?

UEHLINGER: In endemic cretinism the

thyroid gland shows varying degrees of atrophy, fibrosis, nodular formation, progressive degeneration of the follicles and the epithelium. Colloid storage is rare, the colloid basophilic. In sporadic cretinism there is no uniform pattern of the thyroid. Colloid storage may occur.

DARBY: Does anyone know of reports of experimental production of cretinism in animals?

MACKENZIE: If what is meant is small, stunted, blunt-nosed rats, then such animals are born to mothers fed antithyroid drugs. Since one of the most important aspects of cretinism is their intellectual capacity, this is hard to evaluate, although it could be done in experimental animals. But we and many other people have seen young born to animals whose synthesis of thyroxin is essentially nil, and we didn't call them cretins, but everybody else called them cretins. I think this is a matter of semantics.

MATOVINOVIC: In an endemic goiter area the animal companions of man, dogs, horses and cattle have goiters and stillbirths are very common in these animals. Iodine deficiency is a very important problem in the stock-raising industry or in stock farming, because the animals show symptoms and signs which are observed in man.

FOLLIS: To answer Darby's question, one of the early observations by the University of Wisconsin group dealt with the appearance of hairless, cretin-like swine born to mothers who obviously were iodine-deficient; this situation could be cured or prevented by the introduction of iodine into the feed of the sows.

IV. EXPERIMENTAL GOITER

MACKENZIE: The history of man's conquest of organic diseases is the history of their production and study in the laboratory. Until this has been accomplished, the cause of a disease has been rarely understood. Even when astute clinical observation has produced a cure, it has rarely endured until it has been demonstrated under experimental conditions. No better example of this is to be found than the use of iodide in the treatment of endemic goiter, which Coindet introduced with success in 1820 in Geneva. Boussingault is reputed to have said five years later that iodine was the only specific known for goiter (58). Yet, as we all know, the use of iodine was abandoned for nearly a century because the proper dosage had not been established experimentally, and because it seemed improbable that large goiters could be produced by a dietary deficiency of a chemical compound. It was not until Baumann's discovery of the high concentration of iodine in the thyroid gland in 1896, and Marine's analytical studies on the iodine content of goiters at the beginning of this century (59), culminating in his demonstration that iodine prevented the so-called thyroid carcinoma in brook trout (60) and experimental goiter in dogs (61), that the use of iodine for the prevention of goiter in man was reintroduced (62). Even so, the mechanisms underlying the pathology of the thyroid in endemic goiter were poorly understood and controversial. The production and study of experimental goiter has resolved some of these problems and provided us with a probable picture of the development of goiter in man. However, the events leading to the formation of colloid goiter, of nodular goiter, etc., have not yet been elucidated. Until such goiters can be produced and studied experimentally, the underlying cellular pathology, to say nothing of the initial biochemical lesions, will remain matters of speculation.

Before the advent of the antithyroid drugs, the experimental production of goiter was limited to the use of natural food diets. In 1928, Chesney, Clawson and Webster (63–66), while studying experimental syphilis in rabbits, observed that a diet consisting largely of cabbage produced a hyperplastic goiter that could be prevented by iodine. Heat production was reduced and the administration of iodine (8 mg daily) to the goitrous animals resulted in loss of weight and death.

In 1933, Levine, Remington and von Kolnitz (67–69) began an investigation of the goiter produced in rats fed a diet containing 76% of yellow corn. They showed that the size of the goiter was inversely proportional to the iodine intake. In the discussion of their first paper (67) they men-

tioned that the BMR of the unsupplemented animals was −23%. So far as I can tell, this finding was never presented elsewhere in detail. The daily iodine intake required to prevent thyroid enlargement on the corn diet was 1 to 2 μg (68).

Hercus and Purves (70), who did a thorough study of goiter in animals in New Zealand, were unable to produce goiters in rabbits by feeding cabbage, but discovered that rape seed and cabbage seeds were goitrogenic when fed to rats at a 50% level in the diet. The effect of dietary iodine on these goiters was not examined at the time.

Still another vegetable product was shown to be goitrogenic when Sharpless, Pearsons and Prato (71), following the earlier studies of McCarrison, produced thyroid enlargement in rats by feeding a 75% soy bean flour diet. These goiters could be prevented by adding iodide to the diet at twice the minimal protective level reported by Remington (68). Later, Sharpless and Hopson (72) found that the pituitary glands in their goitrous rats contained an increased proportion of basophils and a decreased proportion of eosinophils.

In all of the foregoing experiments the goiters were all of the hyperplastic type and contained little or no colloid. However, only in Remington's animals does the thyroid enlargement appear to have resulted from an absolute deficiency of dietary iodine, and even his work was not without its complications. Within 2 years the maximum thyroid enlargement he could obtain decreased by 50% even though chemical analysis showed that the iodide content of the diet had not changed.

The cabbage diet of Chesney was not deficient in iodine, and was presumed, therefore, to contain a goitrogenic compound. In Marine's opinion (73) this substance suppressed 'tissue oxidation' and thereby increased the demand for thyroxine beyond the level that could be met by the dietary iodine. Administration of iodide increased the rate of thyroxine synthesis.

All of these natural food diets present experimental difficulties. They are chemically and nutritionally ill defined, and they do not promote optimal growth. There is no assurance that their nutritional composition can be reproduced at different times, even in the same laboratory. There is a great need for a highly purified diet which is also iodide-deficient to be used in the study of experimental goiter. Such a diet would provide the basis for new and significant investigations.

However, despite their limitations, the natural food diets did show that the morphological response of the thyroid gland to a variety of conditions was the same. They also demonstrated, to quote Chesney and Webster (65) that, "As Marine has repeatedly shown, iodine in sufficient quantities will protect against all known goitrogenic agents."

Now, I turn to the subject of antithyroid compounds. In 1941 (74) my wife and I, while investigating the possible synthesis of vitamin E by intestinal bacteria, discovered that sulfaguanidine could produce goiters in rats fed a highly purified diet in the absence of any toxic symptoms. Under these circumstances it was possible to eliminate all dietary components as contributory agents and to demonstrate that goiter had been produced for the first time by a pure chemical compound. We at once investigated the chemical configuration responsible for goitrogenic activity and found that it did not reside in either the sulfanilic acid or guanidine part of the molecule. Compounds related to these two parts of sulfaguanidine were therefore tested, and we found to our surprise that both sulfanilamide and thiourea (75) were potent goitrogens. Thus, within a few months, two distinct classes of chemical compounds were discovered to produce large hyperplastic goiters in rats, mice and dogs (fig. 2).

The problem was now to determine the mode of action of these compounds. At the same time, Richter and Clisby (105), also at Johns Hopkins, were studying the toxicity of the bitter-tasting phenylthiocarbamide (phenylthiourea) and observed that its prolonged administration to rats caused a greying of the hair, a fall in body temperature, and the development of hyperplastic goiters. These workers suggested that the goiter was a compensatory hyperthyroidism evoked by the drug's depression of body temperature. The theory that the hyperplastic goiter indicated an increase in

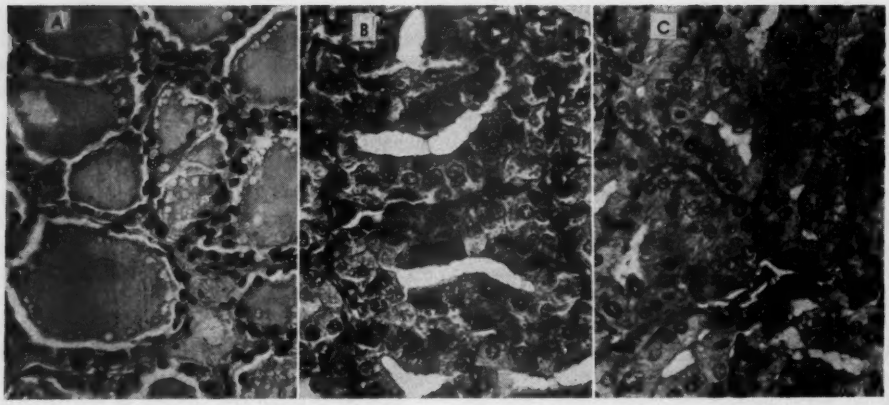


FIG. 2. From left to right: *A*, normal thyroid; *B*, thyroid of young rat fed 2% sulfaguanidine for 14 days, and *C*, thyroid of young rat fed 0.66% thiourea for 21 days. All magnifications × 335.

thyroxine production was still a powerful one.

In contrast to Richter and Clisby, we had been unable to detect a reduction in body temperature or any other toxic symptoms in our animals. Furthermore, we had made the exciting observation that the goiters produced by the sulfonamides and thioureas differed from all previously described experimental goiters in that they were not prevented by even massive doses of iodide. In view of this unique situation, we measured the basal metabolic rates of our goitrous animals and found them to be at or near the thyroidectomy level (76). A critical experiment was therefore called for to determine whether these drugs inhibited thyroxine synthesis, or increased the systemic need for thyroxine beyond the gland's capacity to produce it. It appeared to us that only the thyroidectomized animal, which is highly sensitive to thyroxine, could provide a critical answer to this question. Rats were thyroidectomized, and it was found that the administration of antithyroid drugs to such animals did not inhibit their response to injected thyroxine. We concluded, therefore, that the drugs acted by suppressing the synthesis of thyroid hormone (76), a conclusion that was verified several years later by Keston, Goldsmith, Gordon and Charipper (77), Franklin, Lerner and Chaikoff (78), and Franklin, Chaikoff and Lerner (79) in both *in vivo* and *in vitro* experiments with radioactive iodide.

The foregoing experiments have been described in some detail, for they provided for the first time convincing evidence that the biochemical activity of the thyroid, i.e., thyroxine synthesis, could be divorced completely from the morphological picture of hyperactivity presented by the hyperplastic gland.

The question of the mechanism of thyroid enlargement in animals fed the thioureas or sulfonamides remained to be answered. At an early stage in our experiments we had found that the pituitary glands of rats receiving antithyroid compounds contained large vacuolated basophils and a decreased number of eosinophils (fig. 3). This is a cellular reaction indicative of hyperactivity, and in this case the picture proved to be a correct one. The essential role of thyrotropic hormone in the thyroid hyperplasia of drug-fed animals was demonstrated by removing the pituitary and showing that in these rats, even prolonged administration of antithyroid drugs did not produce goiter. We also showed (76) that the administration of thyroxine to intact drug-treated animals suppressed the thyroid hyperplasia completely (76), (fig. 4).

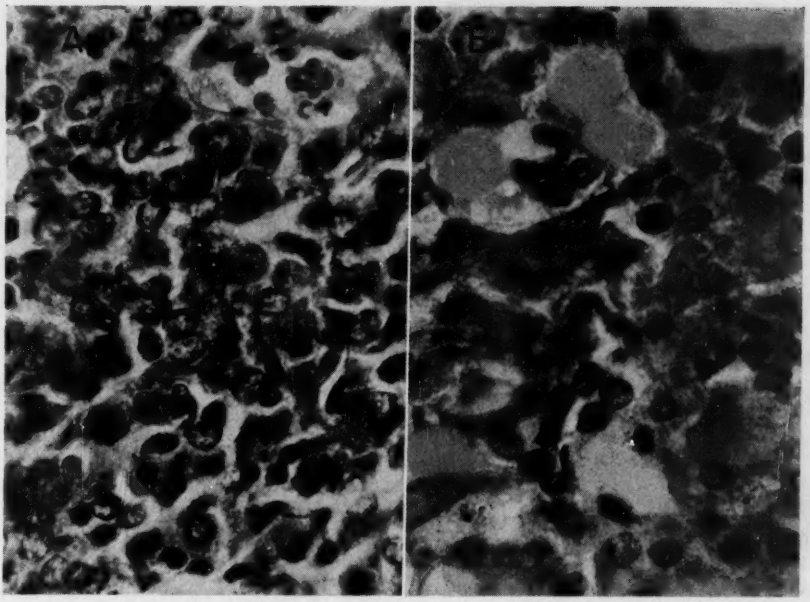


FIG. 3. A, normal pituitary and B, pituitary of young rat fed .75% sulfadiazine for 45 days. × 805.

On the basis of these observations, the production of goiter by antithyroid drugs was formulated as follows (fig. 5). Thyroxine formation is blocked and oxygen consumption falls. This stimulates the production of thyrotropic hormone by the pituitary. The increase in thyrotropin secretion produces hyperplasia, hypertrophy and loss of colloid in the thyroid. Still the thyroid cannot produce thyroxine. Only when the thyroxine is administered or the antithyroid drug withdrawn is this feed-back mechanism restored to normal. While other factors may be involved in the production of goiter, those just described appear to be the primary ones. Indeed, the experiments with antithyroid drugs provided the most convincing evidence for the existence of the thyroid-pituitary axis, and showed that its function did not depend on an absolute or relative deficiency of dietary iodide.

Following the early reports on the goitrogenic activity of sulfaguanidine and phenylthiourea, Astwood, Sullivan, Bissell and Tyslowitz (80) also studied the action of sulfonamides and thiourea on the thyroid, and they reached conclusions similar to ours concerning the inhibition of thyroxine synthesis and the concomitant production of goiter. Moreover, Kennedy, Purves and Griesbach (81, 82) in New Zealand, who were studying rape seed goiter, had found that the thyroid hyperplasia was not prevented by iodide, but was abolished by hypophysectomy. These workers had also observed hyperactive pituitaries in their goitrous animals (83). Later they were to make the important observation that the thyrotropic hormone in the serum of rats fed rape seed had risen to the same level that obtains in thyroidectomy (84). In addition, they showed that the rape seed diet did not sensitize the thyroid to thyrotropin. This is strong additional evidence of the predominant role played by the pituitary in the production of goiter in animals treated with antithyroid drugs.

An intriguing part of the rape seed goiter picture was the inability of Whitehead (85), also at Otaga, to detect any depression in the metabolic rate of rats fed these seeds. This is particularly striking since Purves (86) found that between 2 and 3 μg of thyroxine injected daily was required to suppress the hyperplasia, and this amount approximates the daily output of the rat thyroid.

In 1949 the goitrogenic agent in rape

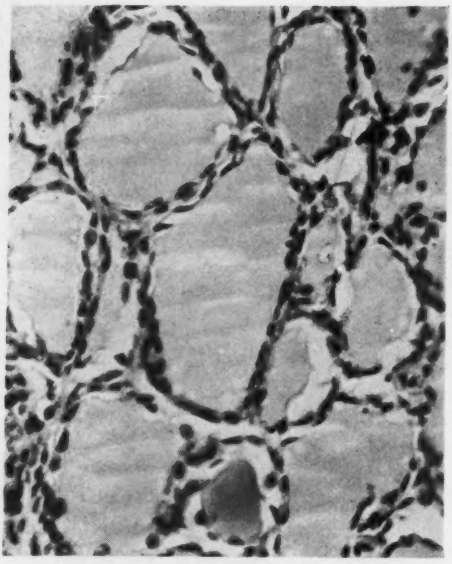


FIG. 4. Thyroid of young rat fed 2% sulfaguanidine and injected with 1 μg thyroxine per 10 gm body weight for 14 days. × 430. This figure is to be compared with fig. 2.

seed was isolated by Astwood, Greer and Ettlinger, and identified as L-5-vinyl-2-thioöxazolidine (87). They also isolated this compound from the seeds of cabbage and kale and from yellow turnips. It shares the configuration —NH—C(=S)—X (where X represents N, O or S) with thiourea and many other antithyroid agents. The extensive investigations of the New Zealand workers with rape seed indicate that thioöxazolidine produces goiters by the same mechanism as the thioureas.

Still another goitrogenic chemical that should be mentioned is thiocyanate. We had found it to be inactive in our early rat experiments (76), although Barker (88) had noted its goitrogenic activity in man in 1936. Astwood (89) resolved this discrepancy by showing that the effect of thiocyanate depended on a low iodide intake, and that its goitrogenic activity was abolished by adding iodide to the diet. Several years later, VanderLaan and VanderLaan (90) and Taurog, Chaikoff and Feller (91) showed that thiocyanate inhibits the iodide-accumulating mechanism of the thyroid. In 1952 Wyngaarden, Wright and Ways (92) made the important discovery that perchlorate, periodate and the other oxyacids of these two halogens have a similar effect on the thyroidial accumulation of the iodide ion.

Entirely aside from the use of antithyroid drugs in the treatment of hyperthyroidism, which was pioneered by Astwood, what do these chemical compounds tell us about the nature of experimental goiter? First, they tell us that the inhibition of thyroxine synthesis results in immediate enlargement of the thyroid in young animals. We could detect a size increase within 2 days of sulfaguanidine feeding in our rats (76). Second, they tell us that a reduction in thyroxine synthesis results in the disappearance of colloid and an increase in height and number of epithelial cells. These changes are so extensive that the lumina of the follicles are practically obliterated (fig. 2). Experiments with antithyroid drugs prove that these changes are not due to iodine deficiency per se. Of course, they also occur when the level of dietary iodide is insufficient for thyroxine synthesis, as in the experiments of Remington and coworkers.[1]

Finally, antithyroid drug experiments have demonstrated that the hyperplastic goiter of thyroxine deficiency is due to pituitary activation. This could not have been achieved in any other way, for only these drugs allow the thyroid, the test organ for thyrotropic hormone, to remain in the body while the pituitary is being activated in the presence of ample dietary iodide. These interrelationships responsible for hyperplastic goiter had not previously been so apparent, for, as Baumann, Metzger and Marine (94) wrote, in 1943, "Many pathologists have found it difficult to accept the view that the hyperplastic thyroids seen in the developmental stages of endemic and experimental goiter produce an insufficient amount of hormone while structurally similar thyroids seen in Graves' disease produce an excess of hormone. In the past we have encountered opposition to the idea that cellular hyperactivity and hypersecretion could be independent. The observations of the Mackenzies that the solfonamides and thiourea can inhibit thyroxine produc-

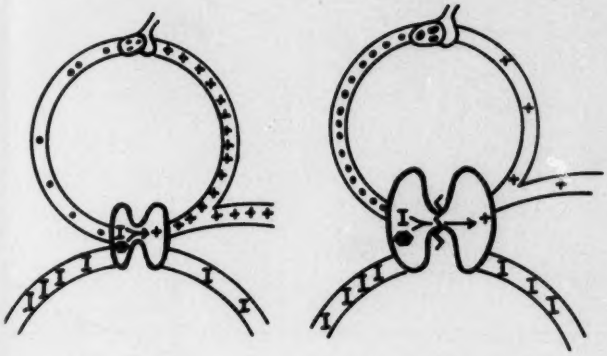


FIG. 5. Schematic diagram of the thyroid-pituitary feedback mechanism elucidated with antithyroid drugs. The normal thyroid is on the left and the hyperplastic thyroid on the right. Open tubes indicate the systemic circulation. Tyrosine is shown by the hexagon and iodide and iodine by I. Thyroid hormone is indicated by + and thyrotropic hormone by ●. The jagged line in the right hand diagram indicates the inhibition of thyroxine synthesis by antithyroid drugs. The development of thyroidectomy cells in the anterior lobe of the pituitary is shown by the enlarged lined ovals.

tion and at the same time cause intense stimulation of thyroid cell growth through the thyrotropic hormone provides proof that intense hyperplasia can be associated with hyposecretion." Earlier, Marine had observed that animals in a region where endemic goiter prevailed had hyperplastic thyroids. He said (60), "Anatomically, a wide range of changes may be present, depending on the species of animal and on the stage (duration) of the disease. In man and fowls one more commonly sees the form characterized by an abundance of colloid material, the so-called cystic or colloid goiter of older writers, while in goiter of dogs, sheep, cattle, pigs, fish, etc., the accumulation of colloid material is seen only in the late, regressive, or quiescent stages;" and, at another time, "Animals with colloid glands are, as a rule, adults."

From the beginning of our investigations we were aware of Marine's view that colloid goiter represented the quiescent form of a previously hyperplastic gland (59) and that the repetition of hyperplasia and regression should eventually produce very large colloid goiters. Accordingly, we put this theory to a test at the first opportunity (95). For this purpose sulfaguanidine was selected as the goitrogenic agent, for, unlike thiourea, its activity is not even slightly reduced by dietary iodide (76). It was found that the repeated administration and withdrawal of sulfaguanidine produced repeated hyperplasia and involution of the thyroid. When the drug was withdrawn after three feeding periods, the glands quickly filled with colloid and the epithelium reverted to the low cuboidal type. After 2–4 weeks without sulfaguanidine, the thyroids of young female rats were still two and a half times the normal size. Under comparable conditions the thyroids of male rats were doubled in size.[1] Whether these colloid-filled goiters would have persisted indefinitely I do not know. Griesbach, Kennedy and Purves (96) conducted similar experiments with their rape seed diet and observed that after five intermittent feeding periods with a final rest period of 3 weeks, the glands were filled with colloid and approximately twice the normal size.[2] In neither of the foregoing experiments on cyclic drug administration were the colloid-filled ascini distended.

We also studied the effect of feeding 2% sulfaguanidine continuously to young rats for a long period of time (95). Such animals were also compared with older animals fed the drug for only several weeks. The results of these experiments are summarized in figure 6. It can be seen that young animals developed hyperplastic thyroids at a much

[1] Money, Rall and Rawson (93) and Axelrad, Leblond and Islen (17) have presented convincing evidence that Remington's diet did not contain goitrogenic compounds. Moreover, the latter investigators produced hyperplastic goiters in mice fed Remington's diet. These goiters were prevented by increasing the iodine content to 12 μg per 100 gm of diet.

[2] Such glands were of course much smaller than the hyperplastic thyroids produced by the continuous feeding of goitrogens.

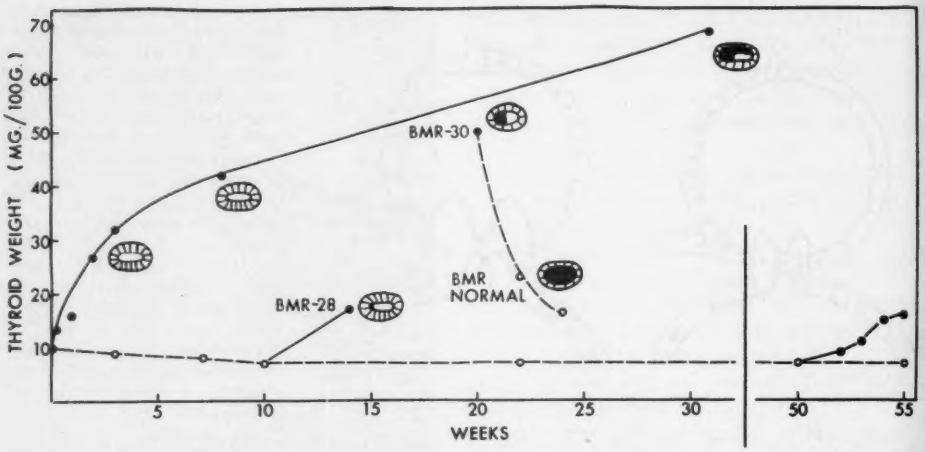


FIG. 6. Effect of 2% sulfaguanidine on the thyroid glands of rats. Solid lines indicate the presence of sulfaguanidine in the diet, broken lines its absence or removal. The oval figures show the height of the thyroid epithelium. The colloid content of the gland is indicated by the black portion of the alveolar space. BMR indicates the basal metabolic rate. All animals were fed the same stock diet and weighed approximately 40 gm at the start of the experiment. Growth was good.

faster rate than older animals. However, thyroxine synthesis was strongly inhibited in the older rats, despite their smaller goiters, as evidenced by the fact that their BMR was at the thyroidectomy level. It seems possible that stimulation of the thyroid by thyrotropic hormone had decreased with age. Nevertheless it was still sufficient, as shown in the top curve, to maintain large glands and to produce a slow increase in size once the goiters were established. That the maintainance of the large goiter was not due to some biochemical change in the thyroid itself was shown by the precipitous fall in thyroid weight, the involution of the gland, and the rise of the BMR to the normal when sulfaguanidine was withdrawn from the diet. Of particular interest was the fact that at 20 weeks on the diet many follicles contained colloid (approximately one-third to half of the normal amount), and cuboidal as well as columnar cells. At 32 weeks most of the epithelium was of the cuboidal type and approximately three-quarters of the follicles were filled with colloid.[3] The size of the goiter was not related to sex in either the very young or old rats. However, at 20 weeks the average thyroid weight (mg per 100 gm body weight) was 34 for males and 57 for females. In the figure these weights were averaged.

These experiments are in harmony with Marine's observations on colloid goiter and age, and they suggest that a controlling factor in the conversion of a hyperplastic goiter to a colloid-type goiter may be a reduction or modification in pituitary activity with age.

Griesbach, Kennedy and Purves (96) and Purves and Griesbach (97) observed that the feeding of rape seed or thiourea to rats for a year and more produced multiple adenomata of the thyroid. The ascini were distended and filled with pale staining colloid and the nuclei were deeply stained. In many instances large colloid lakes contained finger-like projections of columnar cells. During the second year of thiourea treatment 90% of the animals developed these adenometa.

In the tumor-bearing rats killed after 20 months on the experiments, 7 tumors were found that had invaded the walls of adjacent blood vessels and were carcinomatous in appearance. In 2 of these animals metastases were found in the lungs (98). Solid cell masses were present in 3 tumors (2 of

[3] In the majority of rats the thyroids corresponded to this average picture although a few animals had relatively small colloid filled goiters while several others had very large hyperplastic glands.

which were malignant) that resembled the 'fetal adenoma' of man. The investigators believed that these growths had been derived from cancerous tissue. When the thiourea was withdrawn from animals with thyroid adenometa the columnar cells became involuted and there was a marked storage of colloid.

Several years ago Sellers, Hill and Lee (99) fed propylthiouracil to 150-gm rats for 15 months and observed the production of small firm nodules in the thyroid in a number of cases. In other cases the thyroid enlargement was diffuse. Both types of gland contained large follicles filled with colloid. Layers of branching columnar cell cords projected into the colloid lakes. However, in some instances the epithelium which projected into the colloid pools was considerably flattened. According to the authors, some areas resembled human papillary cystadenoma. In three instances metastases were found in the lungs.

In the same experiment, desiccated thyroid was fed to one group of experimental animals. Although the oxygen consumption in these animals was only 8% below normal, the thyroids were 17 times larger than the controls and 3–4 times larger than in rats receiving the thiourea derivative alone. However, the spectacular effect of dried thyroid did not modify the thyroid pathology. The glands were still hyperplastic and contained multiple adenoma. The iodine content was 5.6 μg per 100 gm of fresh tissue, as compared to 2.4 μg in rats receiving propylthiouracil alone and 130 μg in control animals which received neither propylthiouracil or thyroid. In addition to its effect on thyroid size, feeding of desiccated thyroid almost completly offset the growth depression produced by the propylthiouracil.

Glock (100) has observed a hyperplastic goiter, containing colloid, in dogs fed thiouracil for 8–14 months. The epithelium was increased in height and projected into the colloid. In many cases the epithelium was several layers thick. Even the alveoli which were not enlarged were filled with colloid. Thyroid size was increased five-fold and the metabolic rate was normal. In many respects the histological picture of the entire gland resembles that seen in the thyroid adenoma, described above, in rats.

The work reported in this section indicates that the antithyroid drugs and experimental iodide deficiency provide the pathologist with a powerful and essentially unlimited tool for exploring thyroid pathology and for elucidating the factors that give rise to the spectrum of cellular pathology seen in the thyroid of man. With these drugs, hyperplasia, which appears thus far to be the starting point of all thyroid pathology, can be produced at will and with every degree of intensity. Prolongation of the hyperplasia, or more properly prolongation of pituitary stimulation, can give rise to colloid-filled goiters, adenoma, nodular goiters and thyroid carcinoma. Experimental methods exist for determining the effect of protein, vitamin and mineral intake on the development of each of these goiters. The effect of thyroxine in subminimal doses can be evaluated. In this connection, I should like to emphasize an important difference that exists between the rat rendered goitrous with drugs and the man with colloid goiter. In the rat the BMR is at the thyroidectomy level; in the man the BMR is presumed to be normal, or nearly so (101). Thus far in rats that have been cycled with antithyroid drugs it has been an all-or-none situation; complete block in thyroxine synthesis versus return to the normal rate of hormone synthesis. Surely these extremes do not always prevail in the world outside of the laboratory, and I doubt that Marine had these extreme fluctuations in mind when he postulated his cycle. Of course, he never said a *cycle* was essential for the production of colloid goiter. What he did say was that colloid goiters were derived from hyperplastic goiters. In any event, with the judicious use of antithyroid drugs and thyroxine (or perhaps diet) we may be able to produce the elusive colloid goiter in the laboratory.

Although thioureas and sulfonamides both block the iodination of tyrosine in the thyroid, dietary iodide has quite different effects on the size and morphology of the goiters produced by these two classes of compounds (102), and this difference might prove to be very helpful in producing experimental goiters of different types. For

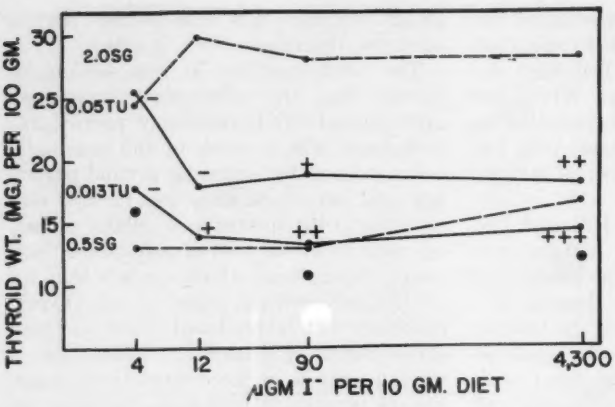


FIG. 7. Differentiation of the goitrogenic effects of thiouracil and sulfaguanidine with dietary iodide. The purified diets, containing 0.05 and 0.013% thiouracil (TU) and 2.0 and 0.5% sulfaguanidine (SG), were fed to immature rats for 2 weeks. The minus signs above the curves indicate the absence of colloid and plus signs the presence of colloid, with 4+ as normal. The unconnected dark circles in the lower portion of the figure show the average thyroid weight of rats fed 2% para-aminobenzoic acid. The thyroid weight of control rats was 10 mg per 100 gm.

example, addition of a small amount of iodide to a purified diet can partially inhibit the goitrogenic activity of thiourea and thiouracil, as evidenced by a reduction in thyroid weight (fig. 7). No such effect is observed with the sulfonamides. Also, while additional increments of dietary iodide do not further reduce the size of the thiourea- or thiouracil-induced goiters, it does reduce the hyperplasia and loss of colloid. With the proper ratio between dietary iodide and thiouracil, one can produce in 14 days an enlarged gland whose follicles are filled with colloid and lined with low cuboidal and squamous epithelium (fig. 8). With sulfonamides, on the other hand, the same level of dietary iodide actually *increases* the size of the goiter and intensifies the hyperplasia and loss of colloid. Prolonging such experiments might produce most interesting results.

Para-aminobenzoic acid, whose goitrogenic activity was discovered by us in 1943, does not respond to iodine like the structurally related sulfonamides, but like thiourea and thiouracil. Iodide minimizes, but does not abolish, its effect on thyroid morphology. These effects of iodide indicate the totally unexpected and unpredictable results that may be obtained when the goiter problem is attacked at the experimental level.

Everything is possible in the laboratory that occurs in nature; and that which is produced in the laboratory is often seen then for the first time in the world at large.

To summarize, goiters have been produced in experimental animals by a variety of natural food diets and by a number of chemical compounds which inhibit the synthesis of thyroid hormone.

A deficiency of thyroxine causes a hyperplasia of the epithelial cells and a loss of colloid from the gland. These reactions depend on the presence of the pituitary gland and are mediated primarily by thyrotropic hormone.

With the prolonged administration of antithyroid compounds, or prolonged iodide deficiency the hyperplastic goiter undergoes a variety of changes which include the reaccumulation of colloid, the development of nodules and adenoma, and the production of thyroid carcinoma (99, 152–154).

Simple colloid goiters characterized by distended follicles and flattened epithelial cells have not yet been produced experimentally. Procedures have been discussed that might produce such goiters.

Antithyroid drugs and iodide deficiency provide the experimental pathologist with powerful tools for investigating the full panorama of cellular pathology that is exhibited by the thyroid gland in human diseases.

FOLLIS: In relation to Mackenzie's presentation, it has always been my impression that Dr. Halsted was the first to produce goiter experimentally by removing a large portion of the thyroid gland from pregnant dogs and finding hyperplastic glands in their offspring (144). Mackenzie tells me

that Dr. Halsted was not able to reproduce this later (145). I imagine that, from what we know today, his original results could now be obtained.

We have encountered goiter in certain studies which we've been carrying on for another purpose. If rats are placed on a diet of corn alone, tremendous enlargement of the thyroid gland takes place. This is not unexpected because, as Mackenzie indicated, the classical diet of Remington contains almost 80% corn. Corn happens to be a grain which is low in iodine. This goiter may be prevented by addition of iodine to the diet. In addition, there is tremendous enlargement of the pituitary, since this gland is really the prime mover. When a control is compared with an animal which has a hyperplastic goiter, the pituitary gland is found to be greatly enlarged. The pituitary shows large numbers of vacuolated cells which are similar to those which are seen when the thyroid gland is removed (150).

Another observation which may throw some light on the relation of iodine to colloid goiter, although this has never been entirely explained, is what happens when iodine is given to a patient who has the hyperplastic goiter of Graves' disease. Rienhoff reported early in the days after iodine began to be used as a therapeutic agent in the preparation of patients for operations for hyperthyroidism that before therapy the gland is extremely hyperplastic, with virtually no colloid. After a course of iodine for 3 weeks all the follicles fill with colloid and the gland becomes less vascular. Concurrently, the patient improves and becomes less of a risk for operation. This suggests that if one gave iodine to the hyperplastic gland of iodine deficiency, it too, might fill up with colloid. This is approximately what Marine postulated (61).

SCRIMSHAW: In questioning the role of iodine in the development of goiter, attention has been called to reports that the iodine of the gland is not reduced, at least per unit of tissue. May we have some comment on the iodine content of goitrous glands and its significance.

STANBURY: We have a few observations on half a dozen or so patients. The amount

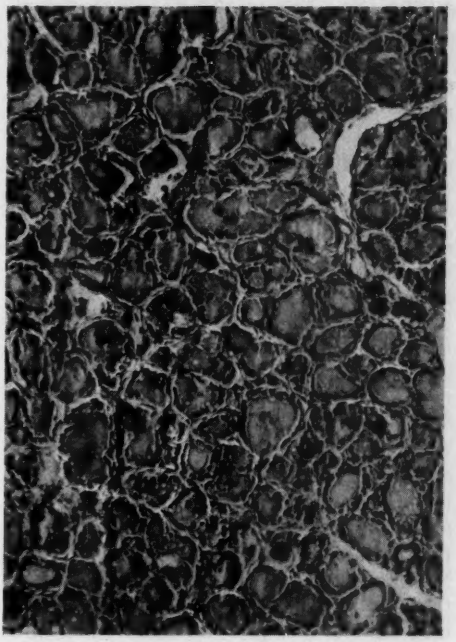


FIG. 8. Thyroid of young rat fed 0.013% thiouracil and 0.05% NaI for 2 weeks (× 130). Such glands were 40% heavier than those of control animals on the same diet with thiouracil omitted.

of iodine in the thyroid gland was calculated on the basis of balance data with I^{127} and I^{131} taken simultaneously. The figures varied in patients with what we presumed to be significant iodine deficiency, from 280 μg in the whole gland to a values of about 18 mg. I have no idea why there is such a wide variance in these glands and their iodine content. Costa in Northern Italy has obtained comparable figures on patients there.

HANDLER: Follis, I didn't understand to what property of corn did you attribute the changes which you saw in the thyroids.

FOLLIS: Iodine deficiency.

HANDLER: May I ask where that corn was grown?

FOLLIS: I can't answer the question as to which state the corn comes from. It was water-ground in Washington. All the studies which have been made on the possibility that corn contains a goitrogen have been negative. Such are studies by Greer (123) and by Leblond (17) and Rawson's group

(93), so I don't think there is too much chance that corn does contain goitrogens.

HANDLER: In that event, do you imply that corn is able selectively to prevent iodine absorption from the soil? If corn, regardless of from whence it comes, is capable of effecting these changes, this would seem to follow.

FOLLIS: The concentration of iodine is such that on this diet you can have iodine deficiency. There is another possibility, that has never been pointedly studied, and this is, what would a lot of essential unsaturated fatty acids do in a diet which contains material which might liberate free iodine?

MACKENZIE: The first studies with corn were made in Charleston, S. C., and with a diet containing 76% yellow corn. Goiters have been produced in Canada with corn and also by Follis with corn. I would think that this in an indication that corn generally is iodine-deficient. Regarding the question that Follis asked about fat; Remington investigated this problem and found that neither cottonseed oil or hydrogenated cotton seed oil had any effect on the production of hyperplastic goiters on the iodide-deficient diet.

SCRIMSHAW: In the course of field trials in rural Guatemala, children with a high incidence of goiter and consuming a predominately corn diet deficient in vitamin A were given a supplement containing 48 gm of either cottonseed oil or lard daily for nearly a year. Neither the addition of vitamin A nor the addition of fat had any effect which we could detect on the size or frequency of goiter in these children.

I suggest that we first try to agree among ourselves as to whether or not goiter has been produced in animals by iodine-deficient diets, and whether goiter thus produced has been cured by adding physiological amounts of iodine into these diets. I think there has been sufficient evidence presented in this discussion to indicate that it has been done.

FOLLIS: It can be said with complete certainty that hyperplastic goiter has been produced in experimental animals, particularly the rat, and that this has been cured with iodine, and that goiter may be prevented by adding iodine to a goitrogenic diet. The experimental production of colloid goiter has yet to be reported.

SCRIMSHAW: The London Study Group in 1952 considered that the level of iodine required in salt to prevent goiter was one part in 100,000 (1). When the 3rd Latin American nutrition conference met in Caracas (4) in 1954, they examined the available data, including the report of the London Conference, and concluded that for Latin America salt should be iodized at a level between 1 part in 20,000 and 1 part in 10,000. Their reasoning stemmed partly from the finding of relatively large amounts of iodine in the crude salts of some of the Latin American countries, levels approaching 1 part of iodine in 100,000 of the salt. This isn't entirely satisfactory information, because this iodine is largely lost over a period of 6–9 months, but the conference concluded that there were probably factors in the Latin American environment which made it unlikely that one part in 100,000 would be as satisfactory as in Europe. They were also influenced by the fact that one part of iodine in 10,000 of salt had been used in Canada and the United States effectively and without difficulty for many years.

In view of this considerable discrepancy, it would be worthwhile to consider any further evidence which there may be for goitrogenic factors operating under natural conditions. Follis' production of hyperplastic goiter in rats with corn diets, and the fact that the people of Mexico, Central America and much of South America who have a high incidence of goiter consume predominately corn diets makes a tempting analogy. However, in Panama where rice replaces corn in the diet, there is as much goiter in some areas as in the highlands of Guatemala with a predominately corn diet. It is also apparent that we are going to have to think of goitrogenic factors in two categories, those which can be overcome by additional iodine and those which cannot. Accordingly, we must not be surprised to discover goitrogenic factors in the environment which can't be overcome by iodine, as already have been found in experimental studies. (14). We have dismissed calcium as a probable factor, and there is no confirmation of the claim that vitamin A deficiency

exerts a goitrogenic influence. Among the factors in plants of the cabbage family some appear to be overcome by iodine, and others not. Obviously we are in a complex and uncertain field, but information or suggestions which we can contribute should be useful.

NADEL: I would like to raise in this connection some of the early reports that unpurified water was involved in certain areas. McCarrison and his group actually fed themselves some of the materials coming from a water supply which led his own group to develop in a short period of time evidence of thyroid enlargement.

SCRIMSHAW: A relation to water supply has been clearly shown on many occasions without elucidation of its cause. Adjacent communities in Central America have been observed, one with a high incidence of goiter and the other with no goiter. In the former, water was obtained from deep wells and in the other from a shallow lake. Recently, I learned about difficulties in the state of Bihar, India, where the change of the water supply to deep wells resulted in the appearance of goiter as a serious public health problem.

PATWARDHAN: McCarrison's earlier observations in an area in the Himalayas indicated that insuring a protected water supply to the population brought about a considerable decrease in 5–7 years in the incidence of goiter in the school children in that area. Previously, he had found that in Gilgit on the northwest frontier of India, although two of three villages received water from the same source, people living at higher levels and nearer the source of water had a considerably lower incidence of goiter than those who lived at lower levels and used water polluted by the residents at higher levels in these hills. McCarrison suggested that polluted water was in some way responsible for the higher incidence of goiter. McCarrison followed up this suggestion by certain experimental work on animals, as a result of which he advanced the hypothesis that a goitrogenic noxa was produced under unsanitary conditions, producing an enlargement of thyroid in his experimental animals. I do not know whether these have been repeated elsewhere and his findings confirmed.

HAZARD: There has been little published recently in regard to this matter of water pollution, and yet it is one of the factors for consideration in regard to goiter production. I recall an early experiment of Marine and Lenhart (60) where tanks of trout were arranged serially, one above the other, so that flow was downward from one tank to the other. The fish in the third and most polluted tank became goitrous. McCarrison's work is certainly stimulating and seems hard to rebut. I don't know how much has been done experimentally to reject or to confirm his observations regarding the inportance of water pollution. There is some recent work in the German literature done by Hettche (103, 104), which I am afraid I haven't read in detail, but which has appeared twice. This has reference to the importance of water contamination as a goitrogenic factor and suggests a substance of the urochrome group may be a causal agent. I would like to hear from someone with more authority than myself in regard to such a goitrogen.

RAMALINGASWAMI: His thesis has been that the substance, whatever its nature is, belongs in the urochrome group, and combines with copper in the serum. This is all I could get in the abstract. I haven't consulted this in the original.

V. Iodine Metabolism

STANBURY: Patients with endemic and non-endemic sporadic goiter constitute by far the majority of those with thyroid disease. It is customary at the present time to accept a deficiency of iodine as the ultimate cause of both sporadic and endemic goiter, whether the deficiency arises from an inadequacy in the diet or because ingress or utilization of iodine is denied the gland by some chemical agent. Obviously such an etiological generalization must be distorted to some degree to cover the facts. Indeed, there is now reason to suspect that sporadic goiter may arise from constitutional disorders which may be entirely unrelated to a deficiency of iodine, as we shall see.

The causes of sporadic and endemic goiter may be classified somewhat arbitrarily into those which are exogenous or dietary in origin, whether they be due to a deficiency of iodine or the presence of a goitrogen in the diet, and those causes which are metabolic or constitutional in nature. In this paper these two classes of causes will be considered in turn, but before doing so, it may be advantageous to review briefly the metabolic circuit of iodine. The subject has recently been reviewed elsewhere in detail (106, 107).

The metabolic circuit of iodine appears in diagrammatic form in figure 9. Iodine may be present in the diet in several forms. For the most part it is reduced to iodide before absorption from the gastrointestinal tract, and appears in the blood as inorganic iodide. A few substances, such as thyroxine, may be absorbed unchanged. The thyroid and the kidney compete for the iodide of the plasma. The relative affinity of these two organs for the available iodide determines the numerical value of the familiar I^{131} uptake test of thyroid function. The normal thyroid achieves a concentration gradient for iodide of 20:1 or more over the plasma. The nature of the concentrating process is not understood. Probably there is loose chemical bonding within the thyroid cells. The iodide in the gland is oxidized to iodine, and exchanges with hydrogen in the 3- and 5- positions of tyrosyl residues in peptide linkage. Monoiodotyrosine is produced before the 5-position is filled to produce diiodotyrosine. These steps, and probably all others in hormone production and secretion, are enzymatically controlled. Mono- and diiodotyrosine, still in peptide linkage, are coupled and an alanine group extruded to form thyroxine or triiodothyronine. Thyroxine and triiodothyronine, together with mono- and diiodotyrosine, are stored as

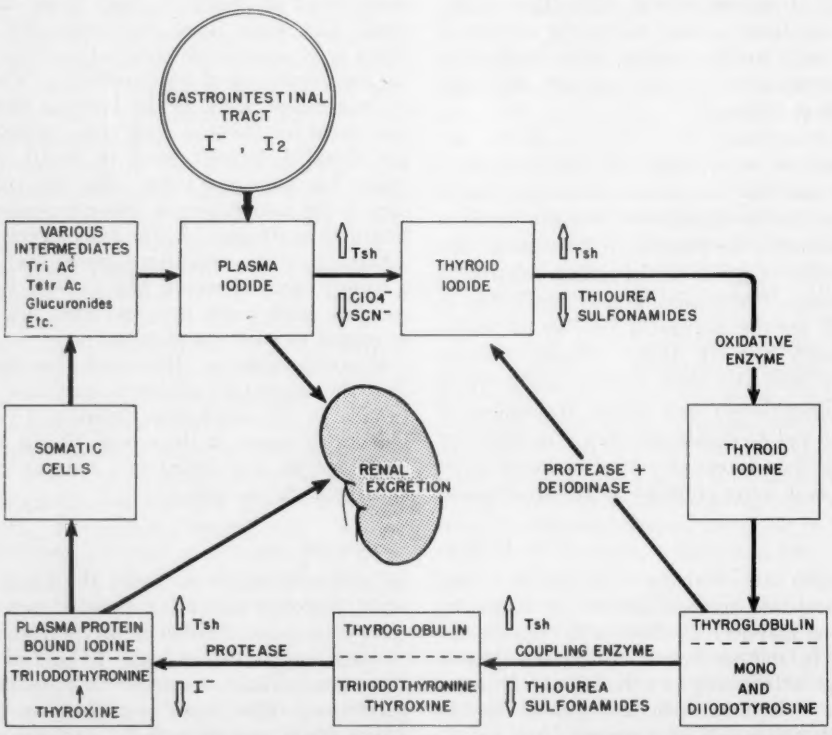


FIG. 9. The metabolic circuit of iodine. Solid arrows indicate the direction of metabolic flow. Open arrows pointing upward indicate stimulation, and those pointing downward indicate inhibition.

thyroglobulin in the colloid of the thyroid follicle. The mechanics of thyroglobulin formation are not understood. For example, it is by no means clear whether the thyroglobulin molecule may be more or less iodinated, depending upon the supply of available iodine, or whether the molecule is irrevocably formed from those iodinated amino acids which are initially available. Little or nothing is known of the sequence of amino acids in the complex thyroglobulin molecule, nor is there any clear concept as to how identifiable a species thyroglobulin, in fact, is. At least three sedimenting peaks have been observed in what appeared to be reasonably pure thyroglobulin (108), but whether more than one of these peaks qualifies as *the* thyroglobulin cannot yet be answered.

Thyroglobulin is degraded by a series of steps which have been studied by Trikojus and his coworkers (109, 110) to yield thyroxine and triiodothyronine to the perfusing blood. In the process, mono- and diiodotyrosine are also released from peptide bondage, but these are rapidly deiodinated within the thyroid cells, and their iodide is reutilized by the gland for a recycling synthesis of thyroglobulin. This deiodinase has only mono- and diiodotyrosine for its substrate.

The thyroid hormones are transported in the peripheral blood largely in close association with a glycoprotein which has an electrophoretic mobility close to α_2-globulin. Thyroxine is tightly bound to this protein, and only a small fraction is found with albumin. Triiodothyronine is less discriminating in its choice of carrier. Both thyroxine and triiodothyronine impinge upon the peripheral cells to have their characteristic metabolic stimulating effect. There is evidence to suggest that this comes about through an uncoupling of phosphorylation from oxidation. There are other theories also for the peripheral effect on tissue metabolism. The iodide produced from hormone degradation in the periphery reenters the iodide pool for reutilization by the gland or for excretion by the kidney. In man, a small part of the circulating iodothyronines are formed into glucuronides for cycling through the liver and gastrointestinal tract. Decarboxylation to thyroacetic acid also takes place. For all practical purposes the only iodine which escapes from the body is in the form of iodide.

Many thoughtful investigators have been unwilling to accept a deficiency of iodine in the daily diet as the exclusive cause of endemic goiter, and with reason. The epidemiological facts which were mustered by Sir Robert McCarrison and others (111, 112) and the historical anecdote which has been the mainstay of Greenwald's argument (113) have counterbalanced the persuasive effectiveness of iodine in preventing endemic goiter. There are now emerging scattered bits of evidence, particularly from the laboratory of Clements in Australia (9, 10) that positive goitrogens in the diets under certain particular circumstances may cause endemic goiter.

Several years ago the author with several of his colleagues made a series of observations on patients with endemic goiter in western Argentina near the foothills of the Andes (55). Approximately 125 patients were studied. Observations included measurements of the protein-bound iodine concentration of the blood and urine and serial recordings of the radioactive iodine which accumulated in the thyroid and was retained there. With rare exceptions these subjects were all clinically in a euthyroid state. Almost all had protein-bound iodine values which were within normal limits, and the mean value was almost identical with that of normal subjects in a nonendemic area. Yet some of these patients had huge goiters, and others had little or no enlargement of the gland. Some had evidence of an intense affinity of the thyroid for iodine, while others did not (fig. 10).

When a correlation was made between the amount of iodine in the diet and the affinity of the thyroid for iodine, a close relationship was found (fig. 11). The primary assumption was made that the iodine which appeared in a 24-hour urine collection was approximately the same as the 24-hour intake of iodine. This value correlated inversely with the I^{131} uptake. Thus, those patients who had small amounts of I^{127} in their diet had a strong affinity for the radioactive iodine. Conversely, those patients who were ingesting an abundance of iodine had no noteworthy increased affinity for iodine by their glands.

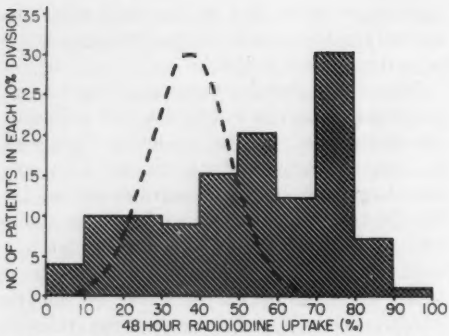


FIG. 10. The 48-hr. radioactive iodine uptake of a group of normal persons in an area of ample iodine intake (dotted line), compared to uptake observed in a group of patients from a goitrous area in western Argentina.

It was apparent that when the daily iodine in the diet fell below 40 μg, it was necessary for demonstrable compensatory mechanisms, as illustrated by increased avidity of the gland for iodine, to take over. These relationships also correlated roughly but reasonally well with the size of the goiter.

Readjustments of thyroid function to a changed level of iodine supply were found to be slow. Groups of patients with high uptake values for I^{131} were given daily supplements of I^{127} and the rate of fall of the radioiodine observed over a period of many weeks. Even when a milligram and a half of iodine was provided daily, the thyroid gland reached a new equilibrium state within the normal limits only after more than 4 weeks. Presumably the same torpid readjustments would obtain for patients removed from an area of iodine abundance to one of deficiency.

The amount of iodine in the glands of these patients varied enormously. Calculations based on balance data indicated values varying from a few hundred micrograms to many milligrams. Iodine content in the gland correlated well with the rate of turnover of iodine by these glands, as would be necessary in order to maintain a euthyroid state.

The efficiency of utilization of a single dose of iodine is illustrated in figure 12. Groups of patients with iodine deficiency were given varying amounts of I^{127} at the same time that they received a tracer dose of I^{131}. There was little difference in the fraction of the dose retained by the thyroid up to about 5 mg, but above this, most of the iodine over 5 mg was wasted through the kidney.

The excessive avidity of the iodine-deficient gland for iodide can be easily inhibited by administration of desiccated thyroid. Thus, even 65 mg of U.S.P. desiccated thyroid daily lowered the I^{131} uptake in most of the patients within 2 weeks. Further inhibition could be accomplished by increasingly large doses. Inhibition of uptake, however, does not necessarily mean that the goiter will disappear, because the long impact of hyperplasia and the accompanying involutionary changes may not resolve when the growth stimulus to the gland is removed.

These observations on the endemic goiter of western Argentina proved to be entirely consistent with a nutritional inadequacy of iodine. No observations were made which were incompatible with this hypothesis. Similar experimental observations have been obtained in patients with endemic goiter of Venezuela (115), Holland (116) and Finland (117). Only Costa (118, 119) in Italy has failed to find an inverse correlation between iodine supply in the diet and affinity of the thyroid for iodine, and no satisfactory alternative hypothesis has been proposed to account for his findings.

In 1831 Boussingault (120) related the endemic goiter of Columbia to the quantity of calcium in the soil. This would be no more than another in a series of possible relationships which have been suggested through the years, were it not for the experimental observations of Thompson (121), Hellwig (122) and of Taylor (18). Rats on an iodine-deficient diet develop goiter. These observers found that if, in addition, calcium was added in excess to the diet, the size of the goiter which was produced was much increased. The mechanism whereby the calcium produces this effect has not been clarified. The findings, however, when taken with the observations that nitrate, perchlorate and thiocyanate all competitively inhibit the uptake of iodine by the thyroid gland, indicate that investigators must be mindful of the possibility that simple inorganic substances may yet prove a significant cause for endemic goiter either by a direct effect

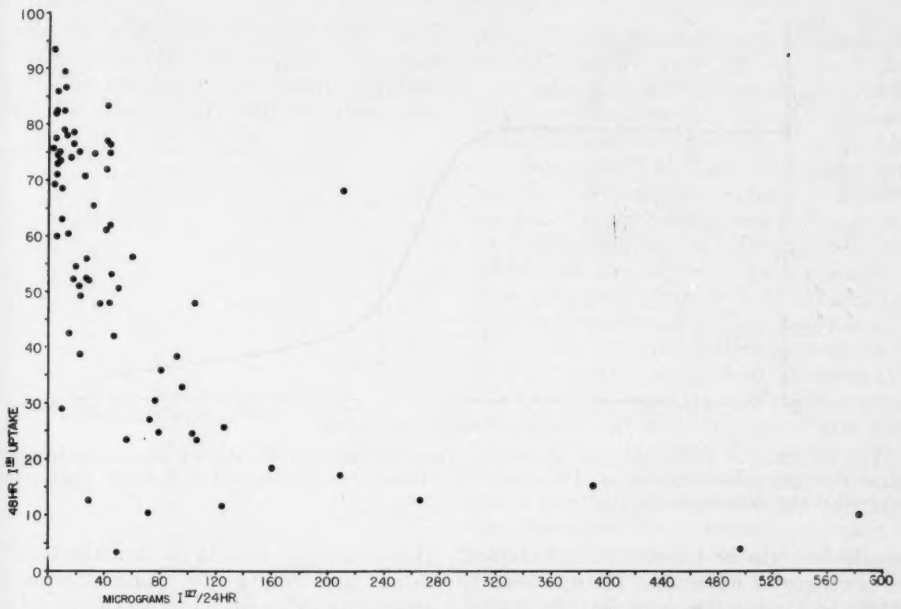


FIG. 11. Comparison of the radioactive iodine uptake (ordinate) with the 24-hr. excretion of I^{127} (abscissa) from a group of patients from western Argentina.

on the thyroid or by competitively inhibiting the retention of iodine by the gland.

Since the studies of Chesney *et al.* (63) and of Mackenzie (74, 76), it has been well known that certain organic chemical substances can cause goiter. Among these substances are thiourea and sulfonamide group of drugs, resorcinol, and a host of others. When confronted with an hyperplastic thyroid of recent origin in a patient without hyperthyroidism, one always entertains the possibility that chance ingestion of a goitrogen may be responsible.

Goitrogenic agents are also present in many kinds of foods (123). One of these, present in cabbage, has been isolated and identified as thioöxazolidone (87). There is little certainty at present, however, that these are responsible or even contribute to to the development of endemic goiter, although the possibility remains.

Clements in Australia (124) found that milk from dairy cows fed upon a member of the genus Brassica, chou-moellier (thousand-headed kale), may, on ingestion, inhibit the accumulation of radioactive iodine in the thyroid gland. He has also observed cyclical development of goiter by children in localized areas of Australia, which seems to be quite independent of the amount of supplementary iodine in the diet. He has also observed goiter in sheep which were forced for market earlier in the season. No goiter developed in the same herd when fed at a later date when the grazing foliage had changed (125).

There seems to be little profit in arguing whether iodine deficiency or goitrogenic agents in the diet are responsible for endemic goiter. It is a demonstrated fact that both these factors may be operative and, indeed, be operative at the same time. It is more to the point to suggest that a given endemic be investigated by the proper and appropriate modern epidemiological and laboratory tools in order that each endemic may be properly defined as to its own nature.

Within the past few years several different types of sporadic goiter have been described, wherein the disease appeared to arise because of a genetically determined constitutional defect in the production of the thyroid hormone (126). If one views the complex sequence of events of hormone synthesis illustrated in figure 9, while mindful of the genetic dependence of the various enzymes involved, then he can visualize that hormone

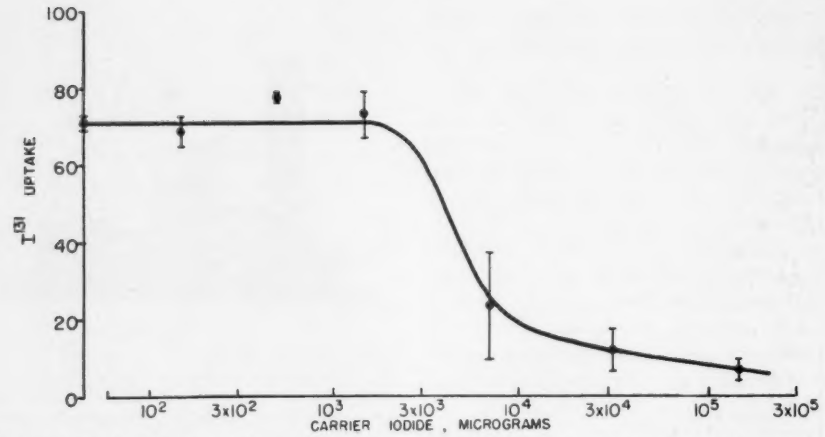


FIG. 12. Effect of stable iodide on uptake of radioactive iodine by the thyroid gland. The two isotopes were given simultaneously and the radioactive iodine uptake measured at 48 hours. Each point represents the mean of several patients.

synthesis could be impeded or interrupted in a variety of ways, that the interruption might be type-specific, and that the disease would follow the usual rules of genetics. It remains to be shown that such factors are operative in any endemic goiter situation, but there is evidence to suggest that they could be so.

In the spring of 1955, a man of 26 presented himself at the Endocrine Department of Professor Querido in Leiden, Holland. He gave a history of a goiter which was first noticed at birth. It had slowly increased in size over the years, and its growth had accelerated when thyroid medication was withdrawn 4 years before admission. Although he was not mentally retarded, a younger brother was both mentally and physically retarded and had also had a goiter since birth. At about the same time, a 12-year-old female was also seen and studied at the same institution. These three patients all shared identical metabolic changes. Upon the administration of radioactive iodine, the isotope was seen to accumulate in the region of the thyroid gland with extraordinary rapidity. The peak uptake was reached within an hour or slightly more at approximately 75% of the administered dose. There was a sharp inflection at the peak, and the labeled iodine of the gland was rapidly lost within the next few hours, so that by 24 hours only 25% remained in the gland. Thereafter, the rate of loss in the gland was much less. At the time the labeled iodine was leaving the gland, increasing amounts of iodinated substances appeared in the blood. On detailed analysis, these proved to be in addition to iodide mono- and diiodotyrosine and two unidentified substances. On further study, one of these proved to be a conjugate of monoiodotyrosine.

Two problems were at once apparent. In the first place, why should these substances be secreted into the peripheral blood? Also, since it had been previously shown that mono- and diiodotyrosine are degraded with considerably rapidity upon intravenous administration, why were these substances not degraded, and why did they appear in the urine?

The answer to the second question was sought first. A number of patients without thyroid disease were given intravenous injections of labeled mono-and diiodotyrosine (MIT, DIT). The rate at which labeled iodinated components appeared in the urine was measured. It was found that after administration of DIT, no more than 4% of the administered dose could be recovered in the urine in an unchanged form. The rest appeared as iodide. After administration of MIT, nothing but iodide appeared in the urine. When identical injections of MIT and DIT were given to the three patients in question, no evidence of deiodinization could be found. The iodinated components which

appeared in the urine were either the parent compound or the unidentified component and the previously mentioned conjugate (fig. 13). Thus, it appeared that these three

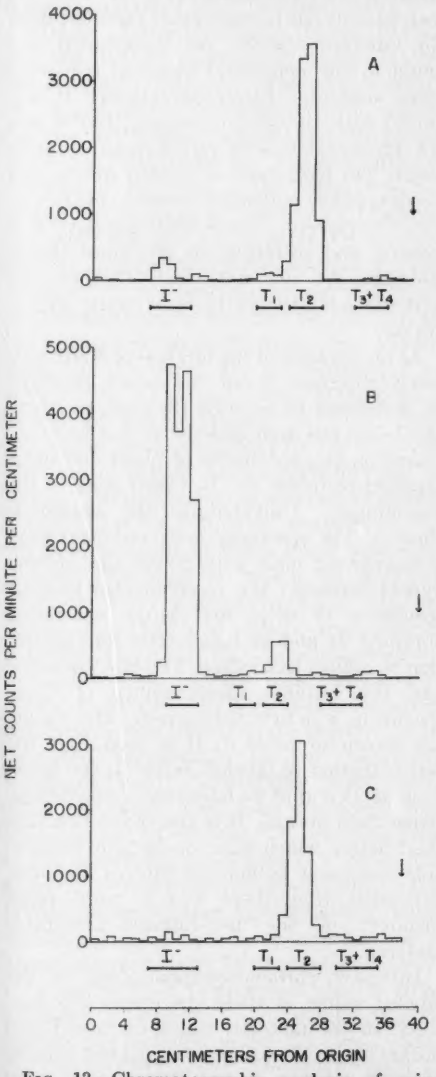


FIG. 13. Chromatographic analysis of urine samples after administration of I^{131}-labeled T-2. Horizontal bars indicate the position of the carrier substances. *A*, chromatogram of administered material; *B*, chromatogram of normal individual after administration of I^{131}-labeled T-2; *C*, chromatogram of urine specimen obtained after similar administration to a patient with cretinism and goiter. I = iodide; T-1 = monoiodotyrosine; T-2 = di-iodotyrosine; T-3 = triiodothyronine; T-4 = thyroxine.

patients lacked the machinery for deiodinating MIT and DIT (127, 128).

In order to determine whether the thyroid glands of these subjects lacked the mono- and diiodotyrosine deiodinase which was described in 1952 by Roche, a series of normal thyroids were obtained from the operating theater of the hospital, and their capacity for deiodinating labeled MIT and DIT was determined. In every case it was possible to show that more than 50% of the labeled substrate was deiodinated. At this point, one of the three patients underwent a thyroidectomy because of tracheal compression. The gland was received, and deiodinization determined. No deiodinization of the substrate DIT was observed. Thus it was shown both *in vitro* and *in vivo* that these patients lacked the capacity to deiodinate mono- and diiodotyrosine. The inference was drawn that they suffered a common genetic abnormality which resulted in a constitutional lack of the tissue deiodinase. Their goiter was assumed to represent a compensatory hyperplasia for the constant loss of iodinated hormone precursors, first from the gland, and then from the body through the kidney (129).

The 12-year-old female just mentioned had a mother who was normal in all respects except for a huge goiter, three maternal aunts with goiter, and a sister with goiter but without retardation or overt hypothyroidism. Limited studies on these persons were permitted (128). It was observed that, while they could deiodinate intravenously administered diiodotyrosine, the quantity of unchanged DIT which appeared in the urine was significantly higher than in a group of normal subjects (table 3). It appeared, therefore, that our three subjects with hypothyroidism and goiter were homozygotes for an autosomal recessive gene, and that the relatives of our 12-year-old female were recessives for the same gene with limited penetrance. McGirr and Hutchison (130, 131) have found an identical metabolic defect in a group of goitrous cretins in Scotland. Their subjects were found in a closely-knit community of itinerant tinkers among whom intermarriage was a commonplace.

Recently the tissue deiodinase for MIT and DIT has been more fully characterized (132, 133). The deiodinase is easily demon-

TABLE 3. PER CENT DIT EXCRETED AS DIT

Patients	Time		
	0–1 hr.	0–2 hr.	0–4 hr.
15 controls	1.8	2.4	2.8
3 goitrous cretins	29.2	46.2	62.3
Relatives of cretin	4.6	6.2	7.1

strable in a simple system consisting of homogenized tissue or tissue fractions, buffer at pH 7.4, thiouracil to prevent re-cycling of iodine, and substrate MIT or DIT. Tissue localization and cofactor requirement are shown in table 4. It can be seen that the enzyme is located in the microsomes and that reduced triphosphopyridine nucleotide is an essential cofactor. The enzyme is thermo-labile, labile to sonic rupture of the microsomes, and inhibited by mercury. We have not been successful in making an acetone powder. If this system is allowed to incubate at 37°C prior to adding substrate, the activity is rapidly lost. As is shown in table 5, however, this is due not to destruction or inhibition of the enzyme, but rather to enzymatic degradation of the required cofactor.

Probably, in time, a goitrogenic defect will be described for each enzyme involved in the fabrication, delivery and utilization of the thyroid hormones. In addition to the deiodinase defect described above, at least three other types are now recognized, although perhaps not quite so precisely defined. Certain cretins have been studied who share the common failing that they are unable to convert iodide to iodine in the gland (134). Iodide accumulates with great rapidity in their glands but never becomes formed into hormones. It can be quantitatively discharged within a matter of minutes by administration of thiocyanate.

Two sisters have been studied in our laboratory, both with retarded mental and physical development, and both with glands with strong affinity for iodide (135). Their defect appeared not to involve the formation of mono- and diiodotyrosine, but rather the conversion of these substances into finished hormones. Without further evidence, it has been assumed that the coupling enzyme is limited or absent in their glands.

Several workers have now reported patients with hypothyroidism and goiter who had an iodinated component in the peripheral blood which was not extractable into acid butanol (126, 136, 137). Normally the iodinated components of the blood are quantitatively extractable into butanol. One such patient has been recently reported from our laboratory (138). An iodoprotein was found in the peripheral blood in relatively large amounts. Electrophoretically it migrated with the serum albumin, but it was not precipitable with anti-human albumin serum. On hydrolysis it yielded mono- and diiodotyrosine and small amounts of thyroxine. The present view is that this patient is making and secreting an abnormal thyroglobulin. We are not clear whether this represents faulty synthesis or faulty degradation.

As an example of the method of study now being employed in our laboratory, it might be of interest to describe the findings which Dr. Leslie DeGroot and the author have obtained on two specimens of blood and urine supplied to us by Dr. B. Courvoisier of the Policlinique, Universitaire de Medecine, Geneva. The specimens were obtained from a 16-year-old male with goiter and lifelong hypothyroidism. We received two 24-hour specimens of urine and serum specimens obtained 24 and 96 hours after administration of radioactive iodine. The labeled iodine and the butanol extractability of these specimens was first determined. The results are shown in table 6. It is seen that the concentration of labeled iodine in the blood both at 24 and at 96 hours was considerably larger than normal. It is also of interest that the fraction which was non-butanol-extractable was lower at 96 hours than at 24 hours, indicating that there was a more rapid turnover of the non-butanol-extractable material.

Intensive chromatographic study of the labeled iodine in these specimens was pursued. In the material which extracted into butanol from the serum, minimal but definite amounts of mono- and diiodotyrosine, in addition to thyroxine and iodide, were found. The detection of MIT and DIT in serum is distinctly abnormal. In the urine specimens, on the other hand, which were applied directly to the paper without prior butanol extraction, small but significant amounts of MIT and DIT were observed, and, in addi-

tion, a highly significant component which, in a butanol-acetic acid solvent system, had an R_F somewhat slower than iodide (fig. 14). This component was not hydrolyzed by β-glucuronidase or by hydrochloric acid.

Studies of the enzymatic degradation of the non-butanol extractable material were not entirely satisfactory. The component in the blood, however, appeared to release mono- and diiodotyrosine and thyroxine upon enzymatic digestion. The non-butanol-extractable material in the blood was not dialyzable, whereas all the labeled iodine in the urine was easily dialyzable. On electrophoretic and curtain electrophoretic analysis of the serum, a strong component was found to correspond in its mobility to albumin. In its salting-out characteristics, the labeled iodine in the blood did not correspond to thyroglobulin but was much more soluble at high salt concentrations.

An hypothesis to account for the observed facts in this patient has been put forward, as follows. The patient has an aberrant synthetic pathway for an iodinated protein akin to thyroglobulin but of much lower molecular weight. This may be a normal pathway which normally is inconsequential, but becomes hypertrophied when the normal pathway is blocked. The abnormal polypeptide is secreted into the blood where it is partly degraded to its component iodinated amino acids, and the residual is excreted as an iodinated polypeptide of low molecular weight and ready dialyzability in the urine. This hypothesis may seem somewhat less than fanciful in view of the recent finding in our laboratory that there is a major low molecular weight component present in the thyroglobulin of several patients with long-standing goiter. This area of investigation is under intensive study at the present time.

In summary it is apparent that a large number of separate and distinguishable diseases are included under the terms endemic and sporadic goiter. The enlargement of the thyroid is only a final common pathway of the disease processes. In all probability, the goiter represents a compensatory effort on the part of the body for inadequate production of thyroid hormones, for if it were a direct stimulating effect on the gland, hyperthyrodism would result.

It is well established that certain endemics of goiter arise because of a profound lack of iodine in the diet. Goitrogenic substances may also be present in the diet. On the other hand, sporadic and familial goiter in certain clean-cut examples may be the result solely of faulty synthesis of thyroid hormones. Probably there are areas of interaction between these general causes of goiter, wherein the presence of a subliminal dietary factor raises a subliminal genetic factor to the level of causing clinical disease. Thus, the impact of the deiodinase defect on the patient either in the homozygous or heterozygous state would probably be dependent largely upon the dietary supply of iodide.

It is a task of the thyroidologist to delimit a particular kind of goiter, either on epidemiological or hereditary grounds, to determine whether there is an exogenous or constitutional cause, or both, and to define at the biochemical level such cause or causes as are manifest. The program is not an easy one. For, as the foregoing has shown, not only are the techniques of the epidemiologist and the geneticist required, but the contribution of the enzyme and the protein chemist will be necessary before the final nature of these diseases is clarified.

OLSON: There are one or two points which occurred to me about the pathogenesis of the biochemical defect in the three goitrous

TABLE 4. COFACTOR REQUIREMENT OF THYROID DEIODINASE

	Substrate	
	3.5 μmoles	16.8 μmoles
	mμmoles iodide formed	mμmoles iodide formed
Microsomes only	0.1	0.1
Soluble fraction only	0.6	1.1
Microsomes + soluble fraction	3.5	6.5
Microsomes + G-6-P	0.1	0.2
Microsomes + glucose-6-phosphate dehydrogenase	0.0	0.0
Microsomes + glucose-6-phosphate dehydrogenase + G-6-P	0.2	0.2
Microsomes + glucose-6-phosphate dehydrogenase + G-6-P + TPN	2.7	5.5
Microsomes + G-6-P + TPN	3.2	5.5

MIT deiodination in mμmoles per hour per 150 mg. thyroid tissue equivalent as affected by various substances. G-6-P, 5 μmoles; TPN, 0.8 mg; glucose-6-phosphate dehydrogenase, 0.5 mg. Total volume, 3 ml. Incubation time, 20 minutes.

TABLE 5. DEGRADATION OF TPNH BY THYROID HOMOGENATE

Preincubation at 37°C	Added at 30 min.	% Substrate Labeled MIT Degraded	
		Added MIT, μg	
		0	5
Mic. + Sup. + TPNH	Mic.	28.4	17.7
Mic. + Sup.	Mic. + TPNH	70.7	52.0
Sup. + TPNH	Mic.	21.2	16.0
Sup.	Mic. + TPNH	58.6	38.4
Mic. + TPNH	Mic.	41.1	29.5
Mic.	Mic. + TPNH	48.9	34.6

TPNH: 5 mμ moles added to each flask. Total volume, 4 ml. Mic.: 0.5 ml microsome suspension. Sup.: 0.5 ml. 10^5 *g* supernatant solution. A small amount of MIT was present in each flask as carrier for the labeled MIT. Substrate MIT added after 30 minutes preincubation. Deiodination stopped after 10 minutes.

cretins with di-iodotyrosinuria which Stanbury studied. As I understand this disease, it is characterized by lack of a deiodinase for di-iodotyrosine. If this is the case, it seems to me that more di-iodotyrosine would be available within the gland for thyroxine synthesis than normally, which is not entirely consistent with the reduced PBI and hypothyroidism found in the illness. One would have to postulate either a lack of the 'coupling enzyme' or some other mechanism for the gland to secrete di-iodotyrosine in order to account for the prompt appearance in the blood and urine of radio iodotyrosine after the administration of iodide-131 as well as the reduced circulating thyroxine.

STANBURY: This has been a puzzling problem to us as well. The only explanation we have to offer is that this is an intensely stimulated gland. We suspect that the earliest products in the process of thyroglobulin synthesis are immediately degraded and extruded by the gland. Accordingly, mono-and/or di-iodotyrosine are secreted almost at once and never hang around long enough to be made into appreciable amounts of thyroxine. As a matter of fact we did find detectable thyroxine in the blood of the patients. The alternative explanation, that there is a coupling defect, is interesting. It may perfectly well be true, but I prefer to make a single diagnosis. It may well be that in some way the de-iodinase and the coupling enzyme are interrelated someway.

MACKENZIE: Perhaps bound tyrosine rather than free tyrosine is iodinated in the thyroid which then might be converted to thyroxin without becoming free tyrosine. Indeed, thyroxin may be synthesized in iodinated casein *in vitro*. The question, then, is: does the de-idonase act only on free iodotyrosines or can it also de-iodinate peptide bound iodotyrosine?

STANBURY: All the studies that have been done indicate that only the mono-and di-iodotyrosine can act as a substrate for this enzyme. The enzyme is a microsomal enzyme which requires TPNH for its action.

MACKENZIE: In the interesting situation you described, where thyroid hormone is bound to an unusual protein in the blood that cannot be utilized, does the blood remove added thyroxine?

STANBURY: The capacity of the blood of the patient who had the non-butanol-extractable material to bind thyroxine was normal.

HANDLER: I'm not sure I know just what this reaction is. This is a TPNH-dependent enzyme, if I recall correctly, isn't it? What are the reaction products?

STANBURY: We checked this with carbon-labeled mono- and di-iodotyrosine and we found only iodide and tyrosine as products.

HANDLER: Is there an O_2 requirement at the same time?

STANBURY: No. We've done this under exhaustive conditions of anaerobiasis. It goes on perfectly well under nitrogen.

HANDLER: The TPNH then is oxidized and you get TPN+.

STANBURY: If the reaction mixture is

TABLE 6. LABELED IODINE IN SERUM AND URINE OF A PATIENT WITH CONGENITAL GOITER

Specimen	%/Liter	%/24 Hr.	% PBI^{131}/Liter	% PBI^{131} Nonextractable
24-hr. serum	.65		.48	72.9
96-hr. serum	.81		.69	56.6
0- to 24-hr. urine		10.5		
72- to 96-hr. urine		3.1		23.2 (% total I^{131})

allowed to stand at 37°C for 30 minutes, the TPNH slowly disappears. The optical density doesn't disappear, but the recoverable TPN from the TPNH, using glutathione reductase, does disappear. The TPNH is split down the middle. This has nothing to do with the iodinization at all.

SCRIMSHAW: Will Stanbury comment on the significance of protein-bound iodine levels in areas where endemic goiter is prevalent. In the observations from Central America we have found lowered levels of protein-bound iodine, but equally so in children with and without goiter. With the administration of iodide or iodate for periods of time, there is a doubling or more of the PBI levels, but, of course, the significance of this is uncertain (fig. 15). What value do PBI determinations have in endemic goiter areas?

STANBURY: I suppose it depends on how severe the endemic is as to whether it would have any value, but the endemic is going to have to be severe before determination of PBI would have much value. The reason I say this is that in our own studies in Argentina on a group of 125 patients the mean PBI was almost identical with the mean PBI of a group of patients in Boston, the determinations all being done in the same laboratory. Now the same thing has been true in Holland in Querido's patients with endemic goiter—which does exist there. Lamberg's group in Finland has found much the same thing, and I think Costa has found that his patients with endemic goiter in Italy have normal PBI's. I don't know what

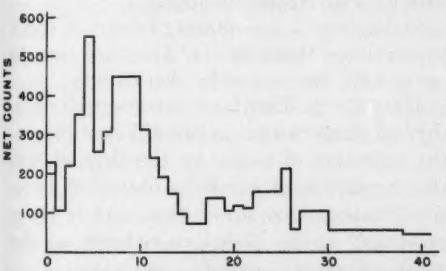


FIG. 14. Chromatogram from the urine of a patient with goiter. Note the zone of labeled iodine preceding the iodide spot and the minimal amounts of MIT and DIT. I = iodide; MIT = monoiodotyrosine, DIT = di-iodotyrosine.

WEEKLY TREATMENT	ORIGINAL STATUS	N°	TOTAL PROTEIN		P.B.I.	
			gms.%	s.	mcgs.%	s.
PLACEBO	GOITER	12	6.60	0.20	2.80	1.23
	NO GOITER	12	6.68	0.37	2.56	1.21
0.5 mgs. KI	GOITER	14	6.55	0.49	5.46	1.40
	NO GOITER	12	6.59	0.41	4.80	0.95
0.5 mgs. KIO_3	GOITER	15	6.65	0.25	4.93	0.98
	NO GOITER	12	6.44	0.40	5.03	1.14

N.F. MCAP-59

FIG. 15. Serum total protein and protein-bound iodine levels in Guatemala after 32 weeks.

the significance of this means. Perhaps excellent compensation, at a goitrous level.

SCRIMSHAW: Would you like to indicate what you consider the range for normal PBI to be, and also when there is an increase in PBI with iodine administration. Do you feel that this does indicate an increase in circulating hormone, or iodine associated with nonactive protein?

STANBURY: The mean PBI in our laboratory, and this is something that varies to a degree from laboratory to laboratory, runs from about 3.5 to 4 μg % at the lower limit and 7.5 to 8, perhaps 9 at the upper limit. Why the PBI goes up on the administration of iodide could be that your technician is not washing the protein precipitate carefully enough to get rid of all the iodide and simply is carrying it along. Danowski at Olson's institution showed some years ago that if one gives large amounts of iodide, enough to keep the blood concentration above 25 μg per 100 ml, he gets some sort of non-calorigenic iodinated protein. In the ordinary administration of iodine I wouldn't suspect one would get any increase in real PBI.

SCRIMSHAW: One reason for asking the question, of course, is the interpretation of values which have been encountered. Among children in rural Guatemala, PBI's are well below levels generally considered as normal. They increase to the normal range, and the fact that controls and experimentals are run at the same time intermixed is, I think, sufficient protection against contamination of glassware and laboratory error.

HANDLER: If you use I^{131}-thyroxine or tri-iodothyroxine—is there any evidence anywhere that this penetrates into the central nervous system?

STANBURY: Yes, if one gives labeled tri-iodothyronine, he can show labeled

iodide in the hypothalamus (151). Handler was going to tell us, how it is that thyroxine acts on the peripheral cells.

HANDLER: The available information is at a rather gross level. The problem which biochemists have thought they could tackle with some success is the effect of thyroid hormone on the rate of oxygen consumption. The fact that dinitrophenol uncouples oxidative phosphorylation led Martius, and, after him, quite a number of people in this country, to attempt studies on the effect of thyroxin on oxidative phosphorylation in rat liver and kidney, with considerable success. In the presence of relatively large quantities of thyroxine such uncoupling does occur, and were it also to occur *in vivo*, there is no doubt that oxygen consumption would rise, since the oxygen which is consumed in uncoupled oxidations is completely useless physiologically. There is no brake on the burning of glucose, and it can increase markedly since only the coupled oxidations lead to ATP formation. However, the mechanism has not turned out to resemble that after which it was originally modeled. The dinitrophenol effect is undoubtedly at an enzymatic level, whereas the effect of thyroxine relates to the physical state of the mitochondria which swell somewhat as they do in non-isotonic media. This has led to some suspicion as to whether this represents a physiological action of the hormone. It isn't known whether this is a reversible phenomenon. Perhaps a bit more unsettling is the fact that the low BMR of the hypothyroid individual cannot be accounted for in this way, since if one were to extend this concept of the effect of thyroxine on the P:O ratio for oxidative phosphorylation, it follows that what we have called normal mitochondria are not completely coupled, and that the normal P:O ratio, instead of being 3 for the oxidation of DPMH, for example, should be a number like 4 or 5, or something more than what we do observe. This has no basis in any fact that we are aware of, and most of us are rather suspicious of this demonstration. There are, of course, the other observations, which relate to the effect of thyroid hormone in driving amino acids into cells so that protein synthesis is accelerated, perhaps by the simple concentration of amino acids, perhaps by something more complex that we don't know anything about. But this is yet another instance in which biochemists have been stymied. For a long time we have thought that all hormones can do presumably would be to affect the rate of an already catalyzed enzymatic reaction, either by inhibiting it, or by inhibiting some other inhibitor. In point of fact, however, these hopes have never been borne out, with the exception of the effect of estradiol in a transhydrogenase system. In all other cases, in order to observe a biochemical effect of a hormone, you have to use something which is at least as complex as a whole cell. And for the biochemist this is not a happy situation.

OLSON: I'm pleased to see Handler's skepticism in regard to some of the *in vitro* studies showing decreased oxidative phosphorylation with added thyroxin. I should like to mention the fact that in some of our studies of exogenous hyperthyrodism in dogs (139) in which we obtained all the clinical signs associated with this disease in man, including increased cardiac output and work, we found no decrease in any of the high energy phosphate compounds (ATP and CP) in the myocardium. Now although this is steady state measurement, in view of the increased work-load of the heart, it may have some significance in denying the postulate of uncoupling based on *in vitro* studies. I would like to hear Stanbury comment about the very low PBI values seen in Guatemala children, with and without goiter. These values we usually associate with hypothyroidism in adults.

STANBURY: I am afraid, Olson, I can't comment on those values. They are new to me and I'd like to ponder them a bit.

MACKENZIE: Thus far we've seen that antithyroid drugs can act in two different places: the collection of iodide by the thyroid and the conversion of iodide to iodinated tyrosine. Stanbury has shown that both of these processes can be absent or reduced as the result of genetic defects, and even more, that two other mechanisms involved in the synthesis, secretion and utilization of thyroid hormone, may be missing because of other genetic defects.

VI. Pathogenesis

Hazard: Since the items that have been considered in these discussions include so many points concerning pathogenesis, I will attempt to do little more than to summarize certain of the areas and perhaps reemphasize some of the statements that were made.

The world-wide distribution of this particular disorder is well known to all of you. A world map of endemic goiter would show a more universal distribution band with many relatively small foci of incidence. In certain parts of the world only a few miles or so of an area may be involved, with adjoining regions showing little or no involvement. The need for explanation of these variances has brought realization of the several problems regarding pathogenesis, the complete answers to which are not known. We will consider certain of the factors later. The need for such an inquiry into endemic goiter as has taken place here is indicated by the discussion which has just preceded this concerning the newer knowledge of metabolic pathways within the thyroid of the goitrous cretin, a problem with which Stanbury particularly has been concerned. Those studies indicate a need for considering the possibility of a defect in the formation of thyroid hormone in certain people with endemic goiter. This may not be the sole cause but, in the presence of lowered iodine consumption, may cause a reduction in the amount of hormone formed. Another point of information which indicates it is important to reconsider this problem is the known existence of goitrogenic substances such as were described so well this morning by Mackenzie in the review of his pioneer work on the sulfanamides and thioureas as goitrogens, and the discussion that followed. The ability of such substances to cause a cessation or a reduction in the amount of thyroid hormone formed provides the altered physiologic setup necessary to effect an alteration in the structure of the thyroid gland. The basic consideration has been previously discussed. I refer to the feedback mechanism regarding thyroid hormone production and thyrotropin release and into which I am sure there is no need for further immediate inquiry. Why there is a lack of thyroid hormone and a consequent alteration in this normal mechanism is a point of present major consideration. The result of this abnormality is demonstrated by certain pathologic manifestations in the gland itself. The early work of Marine quite well demonstrated the thyroidal alterations in the presence of an insufficient amount of iodine, first evidenced by hyperplasia, then as colloid distension with some foci of hyperplasia, and finally with repetition of the cycle, the production of nodular glands. In actual experience the diffuse goiters of endemic type seen in human material are in the colloid phase with sparse if any focal hyperplasia. What is generally encountered, however, is the nodular phase of this disorder, which today is infrequent in any magnitude as a major manifestation in the U. S. A. This indicates one quite important thing—the presence of a nodular swelling in an individual who is youthful, certainly in a child or young adult, in most areas of the United States, is indicative of a neoplastic transformation rather than some form of simple goiter. An interesting illustration of the shift of the type-incidence of nodular change is illustrated in a paper that appeared from the Mayo Clinic several years ago discussing nodular thyroid in children (140). Up to about the mid-30's the nodular change was most often associated with nodular goiter, but in recent years it has much more frequently been a true neoplasm, either adenoma or carcinoma. In other words, there has been a change from the other experience regarding pathological aspects of nodular thyroidal lesions in young people, in that the changes are more prone to be neoplastic. I would like to make one point in regard to diffuse swellings of the thyroid. For a long time patients about the age of puberty or young adults with diffuse swelling of the thyroid were considered to have diffuse endemic goiter. We now know that in this group of swellings are instances of struma lymphomatosa, a lesion which does not respond to iodine therapy and does usually respond to administration of thyroid hormone. Thus, from the point of differential

diagnosis of thyroidal enlargement there is more to consider than endemic goiter.

I would like briefly to draw a comparison between the pathologic findings as evidence in endemic nodular goiter, and the nodular change evidence in sporadic cretinism. I mentioned this earlier and will merely summarize by stating that the usual endemic nodular goiter is compounded of nodules formed of colloid distended follicles and, at times, microfollicular and fetal types; in the congenital goiter, hyperplasia remains a constant factor both in the structure of nodules as well as in the internodular thyroid. An hyperplastic goiter with hypothyroidism may persist for years with retention of the hyperplastic structure. These circumstances are similar to experimental goiter in which hyperplasia is a prominent feature for months.

I wish now to consider some of the statements regarding specific factors in the pathogenesis of endemic goiter. The spectrum so far as the human is concerned is quite broad, many people of many races are involved and the areas of incidence are world-wide. There is no known feature that distinguishes those who will or will not develop the disorder.

The epidemiological factor of first importance, and it has been discussed frequently today, is that of iodine deficiency of the water, soil or food. And such iodine lack certainly must remain a primary consideration, as it has been for many years. Now there is even more support for this being major among the possibilities, since the effectiveness of iodine prophylaxis in many parts of the world is being established. The importance of such a preventive measure is well indicated by one recent study carried out in Ohio in the same four counties in which a survey was made in 1925 (141). The early survey showed a 32% incidence of goiter in school children; that conducted in similar fashion in 1954 showed the remarkable reduction to 4%. It is interesting to note, however, there is still a 4% incidence, and even in families in which there was reasonable assurance on special inquiry that there had been iodine intake because of the use of iodized salt, still 3.4% of individuals had goiter. This makes it necessary to consider the possibility of there being another feature in some persons in addition to the iodine deficiency. There are several factors that must be considered; first, a goitrogen either acting wholly by itself or partly aided by the iodine lack; second, a metabolic defect, perhaps not as complete as those that Stanbury showed a little while ago in goitrous cretins, but still serving as block to thyroid hormone synthesis to the degree that sufficient hormone is produced to supply the patient under ordinary circumstances, but not sufficient to prevent activation of the feed-back mechanism where there is more than usual need for thyroid hormone. Inquiries in regard to the metabolic pathways, possibly defective in these particular people, would be of interest. And possibly associated with this are genetic features which are a part of the constitutional variation in these individuals. However, I do not know of any work which I can cite with the sincere conviction that it establishes hereditary influences as a factor in producing simple endemic goiter. Relations are certainly not as clear as in sporadic goitrous cretinism. It has been stated that hereditary factors may operate in certain isolated areas of the world, such as in valleys which are geographically blocked off from other areas and where intermarriage may give rise to unusual genetic influences. But these are not as established as those seen in sporadic familial goiter and, furthermore, it has been noted that people can move from one area to another, and either, when young have their goiter improved, or have a goiter develop. So far as the present status of this problem is concerned, iodine lack can be considered the commonest feature, whether it be absolute or relative. As to the latter, there may be an hereditary difference in the capabilities of function of certain thyroids concerning their ability to respond to stressful circumstances.

The factor of contaminated water cannot be entirely dismissed on the basis of published evidence. Hard water as a goitrogen was discussed and there was agreement that it probably is not an important feature. The absolute lack of iodine in the water supply is the important consideration, and in most areas is the primary point of prophylactic attack.

Another feature in regard to the etiology

of this disorder is found in the possible presence of a goitrogen the action of which is either partially affected by the intake of iodine, or in some instances may not be influenced at all. The presence of a goiter due to such a substance does not seem likely in many parts of the world, but there is one area in which it does seem to have particular importance. This is represented in the reports that are coming from Tasmania (142). Certainly Clement's work in regard to this has come forth over a period of years and represents a careful study. One must pay attention to the facts that the occurrence of goiter coincided with an increased distribution of milk to children, that there was an equal sex incidence and young children were particularly affected, and the milk came from cattle whose forage has been extended to a form of Brassica chou moellier. Furthermore, an alteration in the uptake of radioiodine by the thyroid has been demonstrated by administering the milk to volunteers. Thus, Tasmania may be one of the areas in which a goitrogen truly may be of prime importance in the pathogenesis of endemic goiter. I do not know of any other region in which such an agent can be considered so definitely a primary cause for the occurrence of the endemic enlargement of the thyroid.

I would like to make brief mention of the health consequences of endemic goiter. The severity of cretinism in certain endemic areas in Switzerland often has been stressed; with iodine prophylaxis apparently there has been a reduction in the incidence of this disorder. In the United States cretinism has never been a part of endemic goiter. The relation of carcinoma to endemic goiter has been mentioned several times today: it may be important in such an inquiry to also consider the type of carcinoma. I have observed that papillary carcinoma occurred in thyrcids in the presence of nodular change in approximately 25% of the cases, whereas with a nonpapillary type this approximated 40%. The difference may be due in part to age distribution, since papillary carcinomas are prone to appear in a younger age group, though they are not of course so limited. Anyway, for proper consideration of carcinoma-goiter relationships, it is requisite that the type of carcinoma be defined. Hyperthyroidism, as you heard this morning, may be of higher frequency in some areas where there is endemic goiter, whereas in other such regions an association does not appear to exist.

In summary, we may say that the most effective agent for control of endemic goiter is iodine. The use of iodate instead of iodide as a salt additive appears to have advantages for use in the tropics. Improvement of sanitation may be a feature, though we still have incomplete evidence as to the role, if any, of polluted water. In areas of ineffective response to iodine prophylaxis, it might be profitable to make studies with the viewpoints of determination of the presence of a goitrogen or the demonstration of certain abnormal pathways for the production or metabolism of thyroid hormones. One important feature of this meeting is to consider means of furthering our knowledge in regard to this particular disorder; to fully accomplish this experimental studies must be further expanded. It would appear up to now that there has not been experimental production in the animal of a goiter that truly corresponds to endemic goiter in man, but I understand we can hope for such a development. Insofar as the present phase of knowledge is concerned, the major etiological factor to consider is iodine lack, and this should be the first point of attack from the viewpoints of prophylaxis and as a part of experimental investigation; realizing, however, that the basic factor is the defective formation of thyroid hormone.

RAMALINGASWAMI: I'd like to make two points on the excellent summarization by Hazard. One is the changing pattern of nodular goiter in the U. S. For those of us working in very highly endemic areas nodular goiter has a special significance, indicating the severity and the duration of existence of the endemic in the area. When one moves into highly endemic areas, the age incidence of nodular goiter is shifted more and more to the left so that one sees nodular goiter in younger and younger age groups as the intensity of the endemic increases. I'd like Hazard to comment on the changing pattern of nodular goiter in the U. S. A., its tendency to be neoplastic and its relation to radiation injury.

The second point is the question of factors other than iodine deficiency in the produc-

tion of goiter. Since the London Conference we have had only one clear example of a positive goitrogenic factor existing in naturally occurring endemic goiter. Nevertheless, there are a few reports which, although not so conclusive, are expanding the idea that perhaps more of such instances might come to light. For instance, Tolley's (146) work in New Zealand, where for several years iodine prophylaxis using fairly high levels of iodine in salt of 1 in 20,000 has been practiced, yet the reduction in goiter is not commensurate with what was expected. The question is raised whether this situation is similar to that of Tasmania. Then, there is Steyn's (147) work in South Africa. Steyn believes that the presence of excessive fluoride and calcium in the drinking water might be more important factors than iodine deficiency. I do not know how extensive the evidence is in favor of this suggestion. There's one suggestion coming from Buttner (148) of South Africa regarding the possibility that tellurium might be of significance in the causation of goiter. Lastly, there is Pales' (149) excellent work in French West Africa, where the 14th parallel divides an intensely endemic area in the south from the one in the north. The difference here seems to be that in the endemic area the people are eating what is described as a salt rich in potassium which is obtained by incineration of plant material. It has been suggested that this substance might contain some goitrogenic factor. None of these examples are as outstanding as Clement's study, but they do show the interest in this field.

HAZARD: I believe that a lower incidence of nodular goiter in the child is associated with a lessening of the endemic insofar as Ohio is concerned. This is reflected also in the paper I mentioned (141) in which a shift in the ratio of carcinoma to nodular goiter for children began to occur about 1938. In regard to radiation, I suppose you mean roentgen radiation so far as babies are concerned? Certain children who have received radiation before the age of 9 months, usually for an enlarged thymus, have later developed papillary carcinoma of the thyroid and the incidence of tumor in this group has been greater than the incidence in the general child population. The actual relationship to the radiation of the thymic area cannot be positively stated. In the adult, I do not know of a circumstance in which direct radiation of the thyroid has been properly considered associated with the production of carcinoma. Radioiodine administration, so far as I can say, up to this time has not been reported as connected with the occurrence of cancer of the thyroid in the human thyroid, as it has, of course, in the experimental animal.

STANBURY: There is going to be one reported at the goiter meetings in about two weeks.

SCRIMSHAW: It is worthwhile to emphasize Ramalingaswami's statement that the occurrence of nodular goiter varies tremendously from area to area and village to village. The pathologists tend to see in their material simply an orderly progression in the number and size of nodules with age. In a severe endemic area, however, you can go from a village in which half the children have goiter and half of these have nodules to another area where the children have goiter but none of them have nodules. You can enter villages in which you can easily find nodules in preschool children and infants.

Another point in relation to pathogenesis is the rapidity of these changes. Ordinarily field studies aren't conducted in a manner which will reveal how rapidly a simple goiter develops in a child or how rapidly it will regress with iodine administration. We have observed goiter develop during a 12-week school vacation. It would be useful to hear other observations as to how rapidly the endemic type of goiter will develop and regress. I also would like to pose the difficult question of why individuals with endemic goiter in villages where there is a lot of nodular goiter don't ordinarily show altered basal metabolic rates.

MACKENZIE: What evidence do you have that they do not show altered metabolic rates?

SCRIMSHAW: The studies that we quote are neither recent nor extensive (13). I don't believe that any of us here have data on this point; it appears that it should be reinvestigated. It should also be mentioned that the reported iodine levels in salt, upon which the 3rd Latin American Nutrition Conference largely based their conclusion that goitrogenic factors were operating in Latin

America, have proved upon re-examination to be too high. Improved analysis of similar samples still indicate levels of iodine in salt approaching 1 part in 100,000 for some fresh samples (54) but not the 5–7 parts per 100,000 which were assumed at the time of this conference. Furthermore, the iodine in these gradually disappears over a period of months (143, 114).

NADEL: Our discussion has dealt with hyperthyroidism but briefly. I would like to know if there is a separate mechanism or a need for a separate mechanism to explain how iodine can improve thyrotoxicosis clinically, at least temporarily, with a drop in basal metabolic rate, sufficient for the thyroid surgeons to have time to remove the gland.

STANBURY: In iodine deficiency the iodine is supplying a need. In thyrotoxicosis we are concerned with some other mechanism, presumably one of blocking the release of the thyroid hormones from the gland. How this is done remains to be shown. Present evidence suggests that it inactivates TSH in some way.

HAZARD: This is a question which I'm sure is unanswerable for the present time. There is some evidence, of course, that iodine also may block proteolytic enzyme activity and thereby bar release of the thyroid hormone.

MACKENZIE: In pursuit of this question I would like to know if it is true that patients with thyrotoxicosis who are treated once with iodine are generally refractory to iodine thereafter.

STANBURY: I don't think so. Patients become refractory to iodine but many persons with thyrotoxicosis can be carried indefinitely on iodine.

SCRIMSHAW: Throughout all of the discussions there has been reference to the need for research on various aspects of goiter. There was particular emphasis on the need for studying endemic goiter in animals, both on various natural diets and particularly on purified diets. There was a clear indication that we need to try to sort out thyroid changes which are due to iodine deficiency or are peculiar to hypertrophy of the gland from those which are common to thyroid glands in goitrous area. Identification of the different types of thyroid carcinoma to determine whether or not they are related to goiter; further study of the nature and distribution of metabolic defects; investigation of cases of supposed endemic goiter which are refractory to treatment; a vast need for further work in the elucidation of goitrogens; a restudy of the question of basal metabolic rates in endemic areas, have all been mentioned as specific topics for investigation, as well as other items listed by Hazard.

REFERENCES

1. World Health Organization. Report of WHO Study Group on Endemic Goiter. London, England, Dec. 8–12, 1952.
2. Great Britain Medical Research Council, Goiter Subcommittee, *Lancet* 1: 107, 1944.
3. Food and Agriculture Organization of the United Nations. Joint FAO/WHO Expert Committee on Nutrition, Report on the Fourth Session. FAO Nutrition Meetings Report Series No. 9, Rome, Italy, July 1955. WHO Technical Report Series No. 97.
4. Food and Agriculture Organization of the United Nations. Report of the Third Conference on Nutrition Problems in Latin America, Caracas, Venezuela, Oct. 19–28, 1953. FAO Nutrition Meetings Report Series, June 8, 1954.
5. Y LOPEZ, G. J. AND F. M. CAICEDO. *Medicina y Cirugia* (Bogata, Colombia) 16: 357, 1952.
6. STACPOOLE, H. H. *Bull. Wld. Hlth. Org.* 9: 283, 1953.
7. SCRIMSHAW, N. S., A. CABEZAS, F. CASTILLO AND J. MENDEZ. *Lancet* 2: 166, 1953.
8. KELLY, F. C. AND W. W. SNEDDEN. *Bull. Wld. Hlth. Org.* 18: 5, 1958.
9. CLEMENTS, F. W. *Bull. Wld. Hlth. Org.* 18: 175, 1958.
10. CLEMENTS, F. W. *Med. J. Australia* 2: 894, 1954.
11. SOKAL, J. *B., J.A.M.A.* 154: 1321, 1954.
12. LUNDE, G. *Verhber. Internat. Kropfkonf. (1933)* 1935; 2. Konf., p. 585.
13. ELMER, A. W. AND M. SCHEPS. *Acta med. scandinav.* 82: 126, 1934.
14. MURRAY, M. D., J. A. RYLE, B. W. SIMPSON AND D. C. WILSON. Medical Research Council Memorandum No. 18, London, England, 1948.
15. HAUBOLD, H. *Verhandl. Deutsch. Ges. inn. Med.* 57: 112, 1951.
16. HAUBOLD, H. *Milchwissenschaft* 6: 285, 1951.
17. AXELRAD, A. A., C. P. LEBLOND AND H. ISLER. *Endocrinology* 56: 387, 1955.
18. TAYLOR, S. *J. Clin. Endocrinol. & Metab.* 14: 1412, 1954.
19. THOMPSON, J. *Endocrinology* 20: 809, 1936.
20. HELLWIG, C. A. *Arch. Surg.* 40: 98, 1940.
21. National Academy of Sciences–National Research Council. *Recommended Dietary Allowances* (revised 1953). A Report of the Food and Nutrition Board, Publication No. 302, Washington, D. C., 1953.

22. British Medical Association. Report of the Committee on Nutrition, London, England, 1950.
23. JOHNSON, N. *Surg., Gynec. & Obst.* 101: 85, 1955.
24. JOHNSON, N. *Brit. J. Surg.* 42: 586, 1955
25. KIMBALL, O. P. Personal communication, May 1951.
26. PEREZ, C., A. SALAZAR-BALDIOCEDA, O. B. TANDON AND N. S. SCRIMSHAW. *Am. J. Publ. Health* 46: 1283, 1956.
27. KLOEPPEL, F. C. *Beitr. path. Anat.* 49: 579, 1910.
28. ASCHOFF, L. *Lectures on Pathology.* New York; Hoeber, 1924, p. 313.
29. WEGELIN, C. *Wien. Klin. Wchnschr.* 1925, nr. 1.
30. WEGELIN, C. International Kropfkonferenz, Bern 1927.
31. WEGELIN, C. *Aerztl. Monatshefte* 4: 643, 1948.
32. MCGAVACK, T. H. *Am. J. M. Sc.* 230: 15, 1955.
33. HUNZIGER, H. *Der Kropf, eine Anpassung an jodarme.* Bern: Nahrung, 1915.
34. PAULLADA, J. J. *Rev. Invest. Clin., Mexico* 7: 29, 1955.
35. OBERNDORFER, F. *Munch. med. Wchnschr.* 65: 1189, 1918.
36. LAMY, M., M. LAMOTTE, AND S. LAMOTTE-BAREILLON. *Bull. mem. soc. med. hôp. Paris* 62: 435, 1946.
37. HOTTINGER, A., O. GSELL, E. UELLINGER, C. SALZMANN AND A. LABHART. *Hungerkrankheit, Hungerodem, Hungertuberkulose.* Basel: Schwalbe, 1948.
38. LUCIEN, M. *Compt. rend. soc. biol. Paris* 64: 236, 1908.
39. MARFAN, A. B. *Nourrisson* 9: 65, 1921.
40. THOMPSON, R. L. *Am. J. M. Sc.* 134: 562, 1907.
41. NICOLAEFF, L. *Presse med.* 2: 1007, 1923.
42. TROWELL, H. C., J. N. P. DAVIES AND R. F. A. DEAN. *Kwashiorkor.* London: Arnold, 1954.
43. AMBROSIUS, K. *Bol. Med. Hosp. Infa. Mexico* 13: 897, 1956.
44. HULL, O. H. *Arch. Path.* 59: 291, 1955.
45. BEHAR, M., G. ARROYAVE, C. TEJADA, F. VITERI AND N. S. SCRIMSHAW. *Rev. Coleg. Medico Guatemala* VII: 221, 1956.
46. JELLIFFE, D. B. WHO Monogr. Ser. No. 29, 1955, Geneva.
47. HELLWIG, C. A. *Am. J. Clin. Path.* 5: 103, 1935.
48. HAZARD, J. B. AND N. KAUFMAN. *Am. J. Clin. Path.* 22: 860, 1952.
49. RICE, C. O. *Arch. Surg.*, 24: 505, 1932.
50. COFFEY, R. J., W. AMOROSO AND R. MAZZARA. *Surg. Clin. North America* 30: 1693, 1950.
51. SCHLESINGER, M. J., S. L. GARGILL, AND I. H. SAXE, *J.A.M.A.* 110: 1638, 1938.
52. NOLAN, L. E. *Arch. Path.* 25: 1, 1938.
53. WEGELIN, C. *Ein Handbuch der speziellen pathologischen Anatomie u. Histologie*, edited by F. Henke and O. Lubarsch. Berlin: Springer, 1926.
54. ARROYAVE, G., O. PINEDA AND N. S. SCRIMSHAW. *Bull. Wld. Hlth. Org.* 14: 183, 1956.
55. STANBURY, J. B. *et al. Endemic Goiter. The adaptation of man to iodine deficiency.* Cambridge, Mass.: 1954.
56. PREBIG *et al. Hegijena* 7: 307, 1955.
57. APFELBAUM, E. *Maladie de Famine: Recherches cliniques sur la famine executées dans le Ghetto de Varsovie en 1942.* Am. Joint Distribution Committee, Warsaw, 1946; (STEIN, J. AND H. FENIGSTEIN. Anatomie path. de la maladie de famine, p. 21.)
58. MCCOLLUM, E. V. *A History of Nutrition.* Boston: Houghton Mifflin, 1957.
59. MARINE, D. AND W. W. WILLIAMS. *Arch. Int. Med.* 1: 349, 1908.
60. MARINE, D. AND C. H. LENHART. *J. Exper. Med.* 12: 311, 1910.
61. MARINE, D. AND C. H. LENHART. *Arch. Int. Med.* 4: 253, 1909.
62. MARINE, D. AND O. P. KIMBALL. *J. Lab. & Clin. Med.* 3: 40, 1917.
63. CHESNEY, A. M., T. A. CLAWSON AND B. WEBSTER. *Bull. Johns Hopkins Hosp.* 43: 261, 1928.
64. WEBSTER, B., T. A. CLAWSON AND A. M. CHESNEY. *Bull. Johns Hopkins Hosp.* 43: 278, 1928.
65. WEBSTER, B. AND A. M. CHESNEY. *Bull. Johns Hopkins Hosp.* 43: 291, 1928.
66. WEBSTER, B. AND A. M. CHESNEY. *Am. J. Path.* 6: 275, 1930.
67. LEVINE, H., R. E. REMINGTON AND H. VON KOLNITZ. *J. Nutrition* 6: 325, 1933.
68. LEVINE, H., R. E. REMINGTON AND H. VON KOLNITZ. *J. Nutrition* 6: 347, 1933.
69. REMINGTON, R. E. AND H. LEVINE. *J. Nutrition* 11: 343, 1936.
70. HERCUS, C. E., AND H. D. PURVES. *J. Hyg.* 36: 182, 1936.
71. SHARPLESS, G. R., J. PEARSONS AND G. S. PRATO. *J. Nutrition* 17: 545, 1939.
72. SHARPLESS, G. R. AND E. M. HOPSIN. *Endocrinology* 27: 129, 1940.
73. MARINE, D., E. J. BAUMANN AND A. CIPRA. *Proc. Soc. Exper. Biol. & Med.* 26: 822, 1929.
74. MACKENZIE, J. B., C. G. MACKENZIE AND E. V. MCCOLLUM. *Science* 94: 518, 1941.
75. MACKENZIE, J. B. AND C. G. MACKENZIE. *Fed. Proc.* 1: 122, 1942.
76. MACKENZIE, C. G. AND J. B. MACKENZIE. *Endocrinology* 32: 185, 1943.
77. KESTON, A. S., E. D. GOLDSMITH, A. S. GORDON AND H. A. CHARIPPER. *J. Biol. Chem.* 152: 241, 1944.
78. FRANKLIN, A. S., S. R. LERNER AND I. L. CHAIKOFF. *Endocrinology* 34: 265, 1944.
79. FRANKLIN, A. L., I. L. CHAIKOFF AND S. R. LERNER. *J. Biol. Chem.* 153: 151, 1944.
80. ASTWOOD, E. B., J. SULLIVAN, A. BISSELL AND R. TYSLOWITZ. *Endocrinology* 32: 210, 1943.
81. KENNEDY, T. H. AND H. D. PURVES. *Brit. J. Exper. Path.* 22: 241, 1941.

82. Griesbach, W. E., T. H. Kennedy and H. D. Purves. *Brit. J. Exper. Path.* 22: 249, 1941.
83. Griesbach, W. E. *Brit. J. Exper. Path.* 22: 245, 1941.
84. Griesbach, W. E. and H. J. Purves. *Brit. J. Exper. Path.* 24: 174, 1943.
85. Whitehead, D. I. E. *Brit. J. Exper. Path.* 24: 192, 1943.
86. Purves, H. J. *Brit. J. Exper. Path.* 24: 171, 1943.
87. Astwood, E. B., M. A. Greer and M. G. Ettlinger. *J. Biol. Chem.* 181: 121, 1949.
88. Barker, M. H. *J.A.M.A.* 106: 762, 1936.
89. Astwood, E. B. *J. Pharmacol. & Exper. Therap.* 78: 79, 1943.
90. Vanderlaan, J. E. and W. P. Vanderlaan. *Endocrinology* 40: 403, 1947.
91. Taurog, A., I. L. Chaikoff and D. D. Feller. *J. Biol. Chem.* 171: 189, 1947.
92. Wyngaarden, J. B., B. M. Wright and P. Ways. *Endocrinology* 50: 537, 1952.
93. Money, W. L., J. E. Rall and R. W. Rawson *J. Clin. Endocrinol. & Metab.* 12: 1495, 1952.
94. Baumann, E. J., N. Metzger and D. Marine *Endocrinology* 34: 44, 1944.
95. Mackenzie, J. B. and C. G. Mackenzie. *Bull. Johns Hopkins Hosp.* 74: 85, 1944.
96. Griesbach, W. E., T. H. Kennedy and H. D. Purves. *Brit. J. Exper. Path.* 26: 18, 1945.
97. Purves, H. D. and W. E. Griesbach. *Brit. J. Exper. Path.* 28: 46, 1947.
98. Purves, H. D. and W. E. Griesbach. *Brit. J. Exper. Path.* 27: 294, 1946.
99. Sellers, E. A., J. M. Hill and R. B. Lee. *Endocrinology* 52: 188, 1953.
100. Glock, G. E. *J. Endocrinol.* 6: 6, 1949.
101. DuBois, E. F. *Basal Metabolism in Health and Disease* (3rd ed.). Philadelphia: Lea, 1936.
102. Mackenzie, C. G. *Endocrinology* 40: 137, 1947.
103. Hettche, H. O. *Arch. Hyg. Berlin* 140: 79, 1956.
104. Hettche, H. O. *Knle. zbl. allg. Path. Anat.* 95: 187, 1956.
105. Richter, C. P. and K. H. Clisby. *Arch. Path.* 33: 46, 1942.
106. Riggs, D. S. *Pharmacol. Rev.* 4: 284, 1952.
107. Berson, S. A. *Am. J. Med.* 20: 653, 1956.
108. Shulman, S., N. R. Rose and E. Witebsky. *J. Immunol.* 75: 291, 1955.
109. McQuillan, M. T. and V. M. Trikojus. *Australian J. Biol. Sc.*
110. Laver, W. G. and V. M. Trikojus. *Biochim. et biophys. acta* 20: 444, 1956.
111. Sinclair, H. M. *The Work of Sir Robert McCarrison.* London: Faber, 1953.
112. McCarrison, R. *C.R. Conf. Internat. Berne,* 1927, p. 304.
113. Greenwald, I. *Am. J. Clin. Nutrition* 3: 215, 1955.
114. Holman, J. C. M. *Bull. Wld. Hlth. Org.* 9: 231, 1953.
115. Roche, M., F. DeVananzi, J. Vera, E. Coll, M. Spinetti-Berti, J. Mendez-Martinez, A. Gerardi and J. Forero. *J. Clin. Endocrinol. & Metabl.* 17: 99, 1957.
116. Terpstra, J. Thesis, Leiden, 1956.
117. Lamberg, B. A., P. Wahlberg and B. Kuhlback. *Sartryck ur Nordisk Medicine* 55: 354, 1956.
118. Costa, A., F. Cottino, G. C. Ferrara, G. M. Ferraris, G. Fregola and F. Marocco. *Minerva nuclear* 1: 3, 1957.
119. Costa, A., G. Cetini, F. Cottino, G. C. Ferrara, G. M. Ferraris, G. Fregola and F. Marocco. *Rev. lyonnaise méd.* 6: 1127, 1957.
120. Boussingault, *Ann. de chim. et de phys.* 48: 61, 1831.
121. Thompson, J. *Arch. Path.* 16: 211, 1933.
122. Hellwig, C. A. *Arch. Path.* 19: 364, 1935.
123. Greer, M. A. and E. B. Astwood. *Endocrinology* 43: 105, 1948.
124. Clements, F. W. and J. W. Wishart. *Metabolism* 5: 623, 1956.
125. Clements, F. W. Personal communication.
126. Stansbury, J. B. and E. M. McGirr. *Am. J. Med.* 22: 712, 1957.
127. Stansbury, J. B., A. A. H. Kassenaar and J. W. A. Meijer. *J. Clin. Endocrinol. & Metab.* 16: 735, 1956.
128. Stansbury, J. B., J. W. A. Meijer and A. A. H. Kassenaar. *J. Clin. Endocrinol. & Metab.* 16: 848, 1956.
129. Querido, A., Stansbupy, J. B., Kassenaar, A. A. H. and J. W. A. Meijer. *J. Clin. Endocrinol. & Metab.* 16: 1096, 1956.
130. McGirr, E. M., J. H. Hutchison and W. E. Clement. *Lancet* 2: 906, 1956.
131. Hutchison, J. H. and E. M. McGirr. *J. Clin. Endocrinol. & Metab.* 14: 869, 1954.
132. Stanbury, J. B. *J. Biol. Chem.* 228: 801, 1957.
133. Stanbury, J. B. and M. L. Morris. *J. Biol. Chem.* In press.
134. Stanbury, J. B. and A. N. Hedge. *J. Clin. Endocrinol.* 10: 1471, 1950.
135. Stanbury, J. B., K. Ohela and R. Pitt-Rivers. *J. Clin. Endocrinol. & Metab.* 15: 54, 1955.
136. DiGeorge, A. M. and K. E. Pashkis. *J. Clin. Endocrinol. & Metab.* 17: 645, 1957.
137. Werner, S. C., R. J. Block and R. H. Mandl. *J. Clin. Endocrinol. & Metab.* 17: 1141, 1957.
138. DeGroot, L. F., S. Postel, J. Litvak and J. B. Stanbury. *J. Clin. Endocrinol. & Metab.* 18: 158, 1958.
139. Piatnek, D. A. and R. E. Olson. *Fed. Proc.* 15: 145, 1956.
140. Hayles, A. B., R. L. Kennedy, L. B. Woodner and B. M. Blacic. *J. Clin. Endocrinol. & Metab.* 16: 1580, 1956.
141. Hamwi, G. J., A. W. Van Fossen, R. E. Whetstone and I. Williams. *Am. J. Publ. Health* 45: 1344, 1955.
142. Clement, F. W. *M. J. Australia* 2: 645, 1957.

143. Zubiran, S. and M. F. Gomez. *Vitamins and Hormones XI*, 97–132, 1953.
144. Halsted, W. S. *Johns Hopkins Hosp. Rep.* 1: 372, 1896.
145. Halsted, W. S. *Trans. Assoc. Am. Phys.* 28: 127, 1913.
146. Tolley, M. A. T. Ann. Rep. Director-General of Health, New Zealand, 1950–51.
147. Steyn, D. G. *Endemic goiter in the Union of South Africa and some neighboring territories*, Pretoria: 1955.
148. Butner, E. E. *S. African M. J.* 9: 187, 1935.
149. Pales, L. *Bull. méd. Afrique occid. franç.* 7: 7, 1950.
150. Follis, R. H., Jr. *Deficiency Disease*. Springfield: Thomas, 1958.
151. Cournier, R. *Ciba Found. Colloq. Endocrinol.* 10: 21, 1956.
152. Bielschowsky, F. *Brit. J. Cancer* 7: 203, 1953.
153. Axelrad, A. A. and C. P. Leblond. *Cancer* 8: 339, 1955.
154. Money, W. L., P. J. Fitzgerald, J. T. Goodwin and R. W. Rawson. *Cancer* 6: 111, 1953.

HYPOVITAMINOSIS A

I. Introduction

Aykroyd: Gentlemen, I am acting as chairman in this discussion on hypovitaminosis A in my capacity as Director of the Nutrition Division of FAO rather than as an expert on vitamin A deficiency, or an important contributor to research on the subject. With great pleasure, FAO joined with the Pathology Study Section of the National Institutes of Health in sponsoring this symposium. You must forgive me if my introduction is of a somewhat elementary nature and that my ideas, such as they are, are probably often many years out of date. We have, of course, many speakers who are well informed on the subject and will provide recent and important information. The discussions of this particular session will be concerned with the clinical, epidemiological and pathological aspects of vitamin A deficiency.

Vitamin A deficiency is still an important practical problem, like goiter and beriberi, which we have discussed during the last two days. Some of our contributors will tell us that manifestations of vitamin A deficiency are still common in certain parts of the world. Some other classical deficiency states, such as pellagra, are becoming rare in this particular era of the world's history, and the same can be said of beriberi in adults. Few members of this group, I think, will have seen an outspoken case of scruvy, and probably only the older members will know what a severe case of rickets looks like, though rickets and osteomalacia are still found in certain regions.

Vitamin A deficiency appeared early in the history of medicine; I think that in this respect it antedates all the other deficiency diseases. The particular manifestation which receives early mention is night-blindness, or inability to see in a dim light. There are, as we know, references to night-blindness and its treatment by liver in ancient Egyptian and Greek medical literature. Coming to more recent, though not modern, times, I myself was delighted to discover a reference to night-blindness and its relation to faulty diet in the works of Reginald Heber, who was Lord Bishop of Calcutta in the early years of the 19th century. A tour of India was made in 1824–25 as part of his episcopal duties and he records the following, which I am venturing to read to you in full, in the lively memoirs of his travels which he subsequently published. The passage is as follows:

> "In our way back through the town [the town was Chitoor in Rajputana] a man begged of me saying that he was blind. On my calling him, however, he came forward so readily to the torches, and saw, I thought, so clearly that I asked him what he meant by telling me such a lie. He answered that he was nightblind ('rat unda'), and I, not understanding the phrase, and having been a good deal worried during the day with beggars, for the whole fort is a swarm of nothing else, said peevishly, 'darkness is the time for sleep, not for seeing.' The people laughed as at a good thing, but I was much mortified afterwards to find that it was an unfeeling retort. The disease of night-blindness, that is of requiring the full light of day to see, is very common, Dr. Smith said, among the lower classes in India, and to some professions of men, such as soldiers, very inconvenient. The Sepoys ascribe it to bad and insufficient food, and it is said to be always most prevalent in a scarcity. It seems to be the same disorder of the eyes which people are afflicted who live on damaged or inferior rice, in itself a food of very little nourishment, and probably arises from a weakness of the digestive powers. I was grieved to think I had insulted a man who might be in distress, but Dr. Smith comforted me by saying that, even in respect of night-blindness, the man was too alert to be much of a sufferer from the cause which he mentioned" (1).

The interesting point is that at that time (1824) the Sepoys ascribed night-blindness to bad and insufficient food, a phrase which has a curiously modern ring. Possibly we should give credit to this early observation to the Dr. Smith mentioned by Bishop Heber. I have not, however, been able to trace who Dr. Smith was. Or possibly this early reference to vitamin A deficiency

might be credited to the Sepoys themselves. But it was, after all, Bishop Heber who put it on record. He was a most remarkable man, who died at the age of forty-three, probably from heat stroke, in a swimming pool in Trichinopoly in which he had plunged to cool himself off after delivering an impassioned sermon. He is perhaps better known as the author of the familiar hymn 'From Greenland's Icy Mountains' than as a pioneer observer of vitamin A deficiency. As a poet and a hymn-writer he could let himself go on occasion. Some of you may remember the verse in 'From Greenland's Icy Mountains':

> What tho' the spicy breezes
> Blow soft over Ceylon's isle,
> And every prospect pleases
> And only man is vile.

This is said to have been written in revenge for the pilfering of his baggage when it was being landed in a port in Ceylon, which he visited on his way to India.

No aspect of the pathology of vitamin A deficiency has been so intensively studied as the relationship between vitamin A and the retinal system and, in particular, visual purple or rhodopsin. This is a field which has attracted a number of able investigators, among whom Wald and Norton should perhaps be especially mentioned. It seems unlikely that at this symposium we shall be in a position to contribute further to what is already known about the complicated subject. I may, however, draw attention to a few rather elementary points. First, the assessment of dark adaptation has proved of singularly little practical value in detecting early evidence of vitamin A deficiency; I have never quite grasped the reason for this, because such an approach is a rational one, and many competent investigators were excited about it some 20 years ago. Secondly, the occurrence of night-blindness is remarkably irregular in patients who show other signs of vitamin A deficiency. I think Oomen will have something to say on that point. Whether night-blindness is always present in some degree in patients showing other manifestations of vitamin A deficiency, is, of course, extremely difficult to ascertain. Cases of severe vitamin A deficiency are most common in young children in whom night-blindness is not readily observed or detected. I may add a third point about night-blindness. There is some evidence that the degree of exposure to strong light may influence its appearance. This is one of the few points on which I can offer a direct observation. The Newfoundland fishermen with night-blindness, whom I saw many years ago, often developed the condition suddenly after a long sunny day at sea in their fishing boats. They would come home in the late evening and have to be led from the landing stages to their sleeping quarters by mates who, though apparently living on the same diet and equally exposed to sunlight, could find their way up the paths from the shore.

Vitamin A deficiency leads to damage to epithelial tissues, and apparently also to nerves and bony structures. With respect to epithelial damage, lesions of the eye are of the greatest clinical and public health significance. They may range from dryness of the conjunctiva and Bitot's spots to the terrible condition of keratomalacia, in which the cornea seems to melt away and the eye is permanently destroyed. Perhaps others will attempt to sort out some of these eye conditions for us and refer to a certain confusion which at present exists in their nomenclature. A bad case of keratomalacia in a young child is a horrifying sight, though of course, modern treatment with vitamin A concentrates is effective, if given in time. I believe that clinicians will agree that this is one of the emergencies of medicine, like infantile beriberi which we discussed two days ago. A few hours' delay in treatment may make all the difference. Two other points of importance about xerophthalmia and keratomalacia may be mentioned. There is often, as we know, a close association between these signs of vitamin A deficiency and protein malnutrition. A further point is that severe lesions of the eyes ascribable to vitamin A deficiencies do not occur in acute famine.

Lesions of the skin associated with vitamin A deficiency are less serious. The whole story of the relationship between vitamin A deficiency and bone and nerve changes still remains, in my view, rather obscure. It would appear to an outsider that the biochemistry and pathology of eye, skin and

other lesions ascribed to vitamin A are poorly understood. There was a time when vitamin A was called the anti-infective vitamin, and the administration of cod-liver oil or vitamin concentrates was recommended as a means of preventing colds and other infections in subjects of all ages. Since there is an increased demand for vitamin A in periods of metabolic stress, the existence of a high reserve of vitamin A in the liver should be, in a broad sense, useful in the maintenance of health and in the hastening of convalescence from diseases of various kinds. But there is, I think, little or no real evidence of any specific anti-infective action on the part of vitamin A.

Some workers have thought that there is a relationship between stones in the bladder and vitamin A deficiency. My predecessor in Coonoor, Sir Robert McCarrison, was among those who upheld this view. In certain parts of Asia, stone in the bladder is extremely common, affecting principally young male children. Among the places where it used to be common are certain areas in northern India; I have no information about its prevalence in these areas today. Another area is northern Thailand, where some facts about its incidence were gathered by Passmore some years ago. Recently, I have heard that it occurs very frequently in parts of Israel. The original hypothesis, as I recall it, was that vitamin A deficiency caused lesions of the mucous membrane of the bladder, which then became sites for the deposit of calcium oxalate. There is, however, no evidence that sufferers from bladder stone in the areas I have mentioned consume a diet which is exceptionally deficient in vitamin A, or show other signs of vitamin A deficiency in any pronounced degree. In Thailand, Passmore was unable to discover any obvious relationship between bladder stone, dietary deficiency and the diet pattern generally.

We are interested, naturally, in the prevention of vitamin A deficiency. The way to prevent vitamin A deficiency is to increase intake of vitamin A—not a very illuminating statement. Vitamin A itself is found only in foods of animal origin, which are in general scarce and expensive in areas where vitamin A deficiency is prevalent. The richest natural source is, I understand, the livers of bears in the arctic region, which are not generally available in South and East Asia. In these areas the increased production of milk is a very difficult and long-term problem; this is, of course, among the main interests and preoccupations of FAO. But the sources of pro-vitamin A throughout the world are unlimited. Here the problem of the absorption of the carotenes requires further study, as well as the metabolic and pathological factors which may influence the conversion of carotene into vitamin A. The facts available on these subjects are not as extensive as they might be. Years ago it was observed in Madras that cases of severe keratomalacia could be effectively treated by a rich carotene concentrate, namely red palm oil. It is interesting that the process of conversion has not yet been elucidated, in spite of the enormous amount of research done on this problem. At all events, this is a field in which further laboratory investigations are needed. Magnificent work leading to the identification and synthesis of vitamin A has been done and no doubt greater use could be made of synthetic vitamin A and vitamin A concentrates in preventive programs. At the moment, FAO, WHO and UNICEF are interested in the enrichment of skim milk powder with vitamin A. This represents a practical problem which should be solved without difficulty in the near future. Enriched skim milk might be a useful preventive weapon, though of course the proportion of children in need who could be provided with such milk is limited.

Perhaps I may refer here to the observations of de Haas in Java before the Second World War (47). De Haas regarded the sale of sweetened skimmed condensed milk as a principal cause of serious vitamin A deficiency. Mothers would feed their infants on this product, often much diluted, with the idea that they were giving them a highly nutritious food. But there is not much danger that the present large-scale distribution of surplus skim milk powder by UNICEF and other agencies will have a similar effect. The skim milk is for the most part distributed through controlled channels, such as maternal and child welfare centers and schools. It is not exposed for sale on the market in small containers. Moreover, it is the general policy of WHO and

UNICEF to provide whole milk powder to infants under one year of age.

We shall, no doubt, touch, in the course of our discussions, on the question of requirements for vitamin A and for pro-vitamin A which has a vital bearing on the problem of prevention. I once observed a group of children showing evidence of vitamin A deficiency who had an intake of about 1700 IU of vitamin A in the form of carotene, which at least gives an indication of what represents an inadequate intake. The human experiments done in Sheffield in England during the war showed that adults whose livers are well stored with vitamin A can remain in good health without this vitamin in the diet for considerable periods of time (90). On the basis of existing knowledge, it is not too difficult to arrive at reasonable estimates of vitamin A and carotene requirements which allow for a margin of safety, but I personally feel that it is a subject which might well be looked at again.

I am sometimes struck by the volume and elegance of scientific research on nutrition on the one hand, and by the fumbling and clumsy methods which we follow in trying to prevent deficiency diseases and raise levels of nutrition on the other. In the practical sphere, there is usually no easy and stereotyped method of attack. A slow approach to education, demonstration, and agricultural and economic development often seems all we can hope for. The nutrition research worker tends to wonder why his results are not immediately applied in the field and the field workers get annoyed with the idea, sometimes held by laboratory workers, that human beings can be handled like laboratory animals. That is one of the reasons why it is useful to hold a symposium such as this at which people concerned with the development of research can meet others concerned with practical field problems and both can exchange ideas and knowledge.

II. Epidemiology

PATWARDHAN: Information on hypovitaminosis A in India is derived mainly from nutrition surveys and from reported observations from hospitals and clinics. The surveys carried out from 1937 to 1942 in various parts of India showed signs and symptoms attributable to vitamin A deficiency varying between 5 and 10% of the children examined. The incidence varied according to the area surveyed, the highest incidence being found in the south and the lowest in the north. I might also indicate here, although this point might come up later for discussion, that we have observed comparatively little association between phrynoderma and ocular signs of vitamin A deficiency. More recent nutrition surveys, from 1942 to date, reveal that post-war conditions have made little difference in the incidence of vitamin A deficiency in India. In 1941 Kirwan (114) from Calcutta showed that nearly 3% of cases seen in the eye infirmary were attributable to vitamin A deficiency. He estimated the incidence of keratomalacia as being 1 per 10,000 of population in Calcutta, the incidence being highest in children below 5 years of age. Our own experience in Coonoor with patients seen in the clinic run by the Nutrition Research Laboratories reveals that out of over 21,000 cases observed during the past 5 years the incidence of ocular manifestations of vitamin A deficiency is about 1.5%. If the incidence is calculated on the number of nutritional disorders only, it would amount to 29%, which would include cases of varying severity.

Hypovitaminosis A is probably one of the principal causes of preventable blindness in India and in Southeast Asia. We, of course, know that the diets are very low in vitamin A; the principal source of vitamin A is carotene and the carotenoids. The fact that infection and infestation play an important role in the acute manifestation of vitamin A deficiency has been clearly brought out by Ramalingaswami in his description of a few cases of keratomalacia associated with acute disease. That there are other factors involved in the incidence of hypovitaminosis A in its most severe forms have been recently made clear by McLaren, who found that in a region in Orissa State in India and within a distance of 30 miles there was a marked difference in keratomalacia among the population living in the hills and that living

on the plains. There were no marked dietary differences which could account for this variation.

McLAREN: May I preface my remarks on the incidence of hypovitaminosis A in Africa by referring to the work which I did in Orissa, India, over a continuous period of 5 years in a hill tribe area (50). In this time I saw 34 children with keratomalacia, responding rapidly to vitamin A alone, but leaving always considerable permanent corneal damage. Only 4 of these cases were in Khonds, the hill aboriginal tribal people. Although they formed 4 out of every 5 admissions of children to the hospital for all conditions, the remaining 30 cases were Oriyas from the plains. Dietary and clinical surveys showed no essential difference in the vitamin A status of these two peoples. I attributed the relative immunity of the Khond children, at least in part, to the family planning practiced by that tribe, whereby it was very unusual for one child to be born within 3 years or so of the birth of a former child. The Oriyas had no such practice, and the history of early weaning onto an unsuitable diet was obtained for several of these children. It is possible that similar marital taboos in some African tribes may help to prevent hypovitaminosis A. Here I would stress the need for more work by social anthropologists in the field of human malnutrition, that of Freedman in Indonesia for WHO being a very good example of what can be done.

Now turn to Africa. We do know that in tracts of lowland tropical Africa, where red palm oil is consumed, there is no vitamin A deficiency problem of any magnitude. However, in East Africa, where I am working at present, certain parts of West Africa and North Africa, vitamin A deficiency may assume serious proportions, especially where the red palm or other rich sources of the vitamin do not grow well. Nowhere in Africa does the association of hypovitaminosis A with protein malnutrition reach such magnitude as it does in some Asian countries, although such an association has been reported from all quarters of the continent. A recent reminder of the need to constantly consider hypovitaminosis A in connection with infections and infestations comes from West Africa, where it has been shown (112) that the posterior segment lesions of onchocereiasis are most marked in areas where both infestation and hypovitaminosis A coexist, and that considerable improvement in vision may often result after vitamin A therapy.

It may not be out of place to conclude with a reference to a condition which has been described from South Africa by Blumenthal in 1950 (113), under the name malnutritional keratitis. He saw many of such cases in malnourished Bantu children in and around East London. I have also observed this condition in its most typical form, in various parts of Tanganyika and in one place in Kenya. From the information I have from others working longer in the area than myself, it appears to be equally common in the East African Bantu people. This condition consists essentially of a spontaneous prolapse of the iris through one part of the cornea. Usually in the lower half of the cornea peripherally there is, in the full-blown picture, a small black dot which appears to be stuck onto the cornea. This is due to a prolapse of iris through a cornea which shows remarkably little evidence of disease. I emphasize this because workers in other parts of the world who have not seen both keratomalacia and this condition, as I have, call this keratomalacia. Here there is absence of lacrimation and photophobia, no dryness, and very little vascularization, just an isolated giving way of the cornea in one or sometimes more spots with consequent prolapse of the iris. The etiology at the moment is quite obscure—it may not be malnutritional at all, but as far as we know it occurs exclusively in the Bantu.

SCRIMSHAW: In general terms, wherever dietary surveys have been carried out in Latin America, inadequate protein of high biological value has seemed to be the first problem among children, and a deficiency of vitamin A the second. Among adults, the principal dietary deficiency appears to be that of vitamin A. Demonstration of clinical consequences of these low vitamin A intakes is much less certain. There are scattered reports of night-blindness which are not well confirmed. It is frequently reported that signs suggestive of vitamin A deficiency are exceedingly common in any survey, but the examples cited are dryness of the skin,

FIG. 1. Serum vitamin A in kwashiorkor after oral administration of vitamin A palmitate. Administration of a single dose of 75,000 μg vitamin A palmitate by stomach tube produced no response in acute kwashiorkor cases on admission, even when a test meal was given at the end of 2 hours. A marked increase in serum vitamin A was obtained in the same test following 5 days of treatment.

follicular hyperkeratosis, and apparently nonspecific eye changes. Follicular hyperkeratosis is very common in clinical surveys in Latin America, but we have been unable in Central America to demonstrate that this responds even to the prolonged administration of vitamin A. Five thousand units were given daily for 30 weeks, 15,000 units of water-soluble vitamin A with skim milk daily for 36 weeks, and 40,000 units for 14 weeks, without effect. Serum levels of vitamin A and carotene found among school children in lower income groups in four of the Central American countries are given in table 1. While lower than values reported in the United States, the serum vitamin A levels are within the lower limits of normal. Serum carotene values are low, except in Guatemala where yellow rather than white corn is consumed. Under certain circumstances the serum levels of vitamin A of adults in Central America will drop below normal limits. The investigation of a presumed outbreak of night-blindness among adults in Nicaragua disclosed that, due to crop failure, many individuals had been subsisting for several months on a diet almost exclusively of sorghum.

PLATT: I would like to draw attention to the prevalence 20 years ago of vitamin A deficiency in China, and to recall that some of the earliest and best work that was done on the eye was done in Peiping. It is also true in that country that the rice eater gets xerophthalmia. With regard to Africa, one day I was asked to go and see an outbreak of night-blindness in an African boarding school in the middle of Tanganyika. It was very interesting, because when I got there and asked the African head master if he could produce any cases, he said "No." I said "Why not?", and he answered, "Because they've just come back from holiday." They had been decently fed at home, and

when they got to the boarding school, the boys were fed on such a poor diet that they get night-blindness. However, I discovered a few cases of Bitot's spots, which gave me material for photographs for teaching purposes. I'd also like to say that one of the most extreme cases of keratomalacia I've seen was in a toddler brought in with extreme marasmus, who died very shortly after admission. There's some story about acute death in young children with severe keratomalacia, but I'm afraid I can't remember the details.

There's a nice story about the Tesa area in Uganda, where the old ladies in the village get the reputation for being able to foretell when the rains are coming. They stay and putter about the hut all day and are not exposed to bright sunlight; they then have the ability to detect distant, so-called 'summer lightning' which the people who are working out in the fields all day can't do. This gift of divination gives them a credit with the villagers because they know when to start getting ready for planting.

RAMALINGASWAMI: I'd like to raise the question of the relationship of vitamin A deficiency and the incidence of trachoma. There are two statements that are most commonly made in under-developed countries about blindness; one, that the most common cause of blindness is vitamin A deficiency, and the other, that trachoma is the most common cause. I think the experience from different parts of the world regarding the incidence of these two conditions might throw some light on this problem, particularly since they are very much handicapped in the study of trachoma for lack of methods for the cultivation of the virus. If one follows the incidence of vitamin A deficiency in India and that of trachoma, one does not find any close correlation. In fact, what one finds is an inverse relationship. At the age of 15, almost 90% of the population in certain areas in northern India have one or other manifestations of different stages of trachoma, as assessed by competent ophthalmologists. As one moves down from the north to the south, one finds a gradual reduction in the incidence of trachoma and rise in the incidence of vitamin A deficiency. I'd like some comment on this from the other members of this group. The second question has to do with the outbreak of night-blindness to which Scrimshaw referred; moreover, the Chairman in his introductory remarks did say that the studies on dark adaptation have not indicated a close association with vitamin A deficiency. The third point is the vexed question of the relationship between vitamin A deficiency and intestinal parasitism, which is again a problem in epidemiology. There are a lot of reports in the literature of a high incidence of both conditions in groups of people, although I don't think there has been any clear-cut demonstration of a specific relationship between the two. In this connection, the reference McLaren made to Rodger's work in Africa on the relationship between posterior degenerative lesions in the choroid, the pigment cells, and the rod and cone cells of the retina, and onchocereiasis, and the dramatic responses which he obtained with massive doses of vitamin A might be of significance. Rodgers presents the hypothesis which needs, I should think, stronger evidence, that some kind of a toxin is produced by this worm, which interferes with the metabolism of vitamin A.

TABLE 1. BLOOD SERUM VALUES IN CENTRAL AMERICAN SCHOOL CHILDREN

Country	No.	Origin	Vitamin A mcg/%	Carotene mcg/%
Costa Rica	142	Urban	28.4%	75%
Honduras	91	Urban	28.6	57
Guatemala	19	Urban	30.5	157
	232	Rural	24.8	108
Salvador	98	Urban	25.8	85
	84	Rural	19.6	44
Michigan	42	Urban	42	134

SCRIMSHAW: In investigating the episode of night-blindness to which I referred earlier, we were unable to measure dark adaptation. The investigation of dark adaptation in serious epidemic situations is an obvious need. The occurrence in Central America of xerophthalmia with keratomalacia in occasional cases of marasmus, particularly in El Salvador, should be mentioned. Occasional cases of marasmus come into the hospital with keratomalacia and may become blind as a result. Marasmus cases in Central America upon occasion show characteristic Bitot's spots, the only instances in which we have seen this ocular manifestation in the area. We have not observed these conditions in

children with kwashiorkor in either Salvador or the other Central American countries.

PLATT: I just want to say that I think the problem of the cultivation of the trachoma virus has been solved by Dr. F. F. Tang, Director of the Serum and Vaccine Institute in Peking, who may be known to some of you as a former student of Zinsser. My colleague, Professor E. T. C. Spooner of the London School of Hygiene and Tropical Medicine, was given the method of cultivation of the virus, and it was successfully tried by a research unit in the Gambia.

PATWARDHAN: The relationship of night-blindness to hypovitaminosis A has to be interpreted with caution under certain circumstances. I would like to refer here to our experience of a few years ago. We were called on to investigate an outbreak of night-blindness in a village near Madras where night-blindness suddenly appeared in epidemic form. We found that dark adaptation was impaired in an appreciable number of people in that village. We could not measure the vitamin A in blood. In a village close by, the intake of vitamin A calculated from diet surveys was lower than in the one in which the night-blindness was complained of, although the impairment of dark adaptation in the former was negligible. We found also that there was extensive hookworm infestation in both the villages with varying degree of anemia. Somewhat to our surprise, we found a certain relationship between the level of hemoglobin in the blood and the incidence of night-blindness in the two villages, as the following figures should show. The fact which results from this investigation is that in spite of twice the amount of vitamin A intake in the village there was a fairly high incidence of night-blindness, which is more than 40% as compared with the other village in which the vitamin A intake was low and the incidence was about 5%. In subjects with hemoglobin levels below 7.4 gm/100 cc, 66% had impaired dark adaptation; in those between 7.5 and 10.4 gm% the incidence was 46.9%, and for levels of 10.5–14.8% it was 36.6%. We then undertook a therapeutic test. One group received a supplement of iron, another a supplement of vitamin A; a third group received neither. All three groups received 8 ounces of rice daily. After 1 month, improvement had taken place in all three groups. In spite of the fact that their diets were more or less unchanged, increasing their intake of rice brought about a material recovery from the state of night-blindness. This investigation thus showed that all outbreaks of night-blindness are not necessarily attributable to deficiency of vitamin A. One must take into consideration other factors, such as the one which I described, that is, hookworm infestation and the degree of anemia in relation to night-blindness.

MCLAREN: I'd like to emphasize what Patwardhan has already said, that other factors than vitamin A deficiency need to be taken into account in the study of night-blindness. For instance, retinitis pigmentosa and a number of other degenerative conditions affecting the retina may all have this as their presenting symptom. In connection with the measurement of dark adaptation, the Sheffield workers (90) found that in their experimental subjects the earliest changes were detectable by rod scotometry, which is the measurement of the visual fields under the condition of dark adaptation. As far as I know this has never been applied in the field. With regard to the question of trachoma and its possible relationship to malnutrition, with possibly the exception of Australian aborigines, those communities which suffer from trachoma are also malnourished, but this does not mean that there is any causal relationship. The second WHO/FAO joint expert committee on trachoma did come to the conclusion that nutrition was one of a number of factors which did play a part in the etiology of trachoma, although there is no real evidence for this. With regard to Ramalingaswami's point about vitamin A deficiency in trachoma, I think that there is no indication of any association. One difficulty is that trachoma varies tremendously in different parts of the world with regard to its course and severity. In North Africa, for instance, it tends to have a rather rapid course and to produce severe changes, with gross scarring of the cornea and marked impairment of vision, in a few years. Whereas in Indonesia, for example, it tends to take a more mild form, may even spontaneously regress, and is not nearly so important a cause of blindness as vitamin A deficiency.

DARBY: There are one or two points which I think it might be well for us to consider against the background of vitamin A status in the U. S. A. First, 30 or so years ago we had some vitamin A deficiency disease in improperly-fed infants in this country. This has disappeared. In fact, the vitamin A status in this country is very satisfactory. Cases are sufficiently rare that they now warrant reporting when they occur. On the other hand, studies of blood levels and of dark adaptation have probably been more widely made in this country than elsewhere. Within the last few months there have appeared reports which conclude that blood studies of serum level of vitamin A have no usefulness in estimating vitamin A status. This conclusion is based on measurements in normal populations with high intake levels of vitamin A. If one then recalls the discarding of dark adaptation measurements in this country, one again finds that the judgment of 'no value' was based on attempts to measure differences in dark adaptations in individuals who were well nourished by all standards that we now know. It was then concluded that these measurements did not reveal vitamin A deficiency, which the investigators were sure existed in the United States, and that therefore this was not a useful procedure. I think it is time to have a reexamination of some type of measurement of dark adaptation.

Other points should be mentioned in consideration of epidemiology. Rolls (to be published) offers the concept that when a population depends entirely on carotene, the fat content of the diet may be crucial in determining vitamin A activity. This may account for some of the differences in vitamin A symptomology as observed in populations subsisting on essentially the same dietary intake of vitamin A.

Finally, in the same 15 or 20 years we find a most unfortunate error led to the conclusion that over 90% of the American population had eye lesions of avitaminosis A. The error lay in the confusion of Bitot spots with pingueculae. The resulting erroneous conclusions have discredited some valuable methodology. These occurrences should be recognized in evaluation of the literature.

BURGESS: With regard to Patwardhan's experience, I would just like to say that during the war I saw some severely malnourished prisoners who were night-blind. When they were given more rice they recovered.

MACKENZIE: With respect to Dr. Patwardhan's observations on night-blindness, it might be pointed out that a decrease of dark adaptation is the first response, and by far the most sensitive response, to any reduction of oxygenation. This comes perhaps at a simulated altitude of 8,000 feet in a few minutes, whereas any other response to anoxia cannot really be detected at altitudes less than 18,000 feet.

HIGGINSON: In a dietary survey in Johannesburg, the average intake of carotenes expressed as vitamin A was 6,000 IU per man unit per diem. That this expressed accurately the status of the community was indicated by the fact that in livers at autopsy of white and Bantu subjects analyzed chemically, we found the range of vitamin A in both races similar. This method might be utilized for getting accurate statistical assessment of the vitamin A status of a particular community.

SCRIMSHAW: The reason that I took pains to cite the lowered levels of vitamin A under certain circumstances was precisely the claim in the U.S. literature that vitamin A values are not useful in indicating decreased vitamin A nutriture. I'd like to state categorically that in situations where there is a severe enough degree of vitamin A deficiency it is a very useful and simple measure.

III. CLINICAL EXPERIENCE ON HYPOVITAMINOSIS A

OOMEN: Clinical experience is always limited to special observational facilities and environments. So is mine with hypovitaminosis A. I want to concentrate as much as possible on personal experience. Therefore, I think that you ought to obtain some details about the surroundings which have produced my conceptions and figures. My tropical career started in a mountain village on the island of Celebes in Indonesia, where for 8 years I was in charge of a mission hospital. My first patient with xerophthal-

mia went blind before I realized what it was. When the second case came to my polyclinic, I listened to my head nurse, who told me that this was vitamin A deficiency and that I should give cod-liver oil to the child. This child now is a teacher with normal eyesight. In those years I saw 20–30 cases yearly, all small village children. Afterwards I came to work in west Java, a very different region. There the condition was encountered even more frequently; again the patients came from rural backgrounds.

Then came the war, and with it my existence of working as an internee doctor in Japanese civilian camps. We experienced many nutritional problems, but xerophthalmia was not one of them. The internees were white people and small children who subsisted on a diet very much like their present Indonesian counterparts in the low income groups. After the war I was in charge of a large city hospital. Among the specialists available there was an ophthalmologist. There I had extensive clinical facilities for biopsies and for serum determinations. Though the clinical material was different from that of the rural hospitals, the city contributed 30–80 cases of xerophthalmia per year. I always tried not to cling too much to the walls of my hospitals, but to find out as much as possible about the environments of the patients. Later again, as a WHO expert, I had access to the central hospital of Djakarta, a city of about three million inhabitants, where, daily, new patients with xerophthalmia were admitted. I learned much at that time from ophthalmologists at Semarang and Jogja, both cities in central Java. The first area once reported on 2,200 cases within 1 year; in the eye hospital of the latter I scanned 11,000 case histories of xerophthalmia.

In passing, I want to stress that xerophthalmia is present in every region of Indonesia, except possibly in some maize-eating regions. It has been studied in urban as well as in rural populations, at sea level and in the mountains, on small islands and along the big rivers of Borneo. One important dietary factor which is common to all these different living conditions, is that all people are rice-eaters; a second factor is the fact that a small child seldom receives an adequate diet.

No other clinical manifestation either dominates so much the symptomatology of hypovitaminosis A or equals the importance from a public health viewpoint as does xerophthalmia. I don't think that there are examples of the efficacy of a single vitamin which are so striking as the administration of vitamin A in xerophthalmia and night-blindness, except perhaps of B_1 in infantile beriberi. If vitamin A does not help within a few days, I doubt the validity of my diagnosis. Xerophthalmia is a horrible disease. While beriberi has vanished from Indonesia at the mere identification of the vitamin, not much progress has been made in avoiding blindness by xerophthalmia, in the nearly 40 years that have elapsed, since Wille (2) described the condition in 268 cases, aged 1–5 years, from the city of Semarang.

The nature of xerophthalmia: What is xerophthalmia? It is seen in the too, too frequent case in which a mother, after long hesitation, ventures to the doctor, because her small child did not open his eyes for 10 days. The doctor then finds two corneas, irretrievably gone. But xerophthalmia is also seen in the case of the schoolchild, who comes to the doctor with a big contusion on his forehead. He ran into a pole the preceding evening at dusk, because he is night-blind, and he has Bitot spots on his eyes.

Clinically, xerophthalmia is an affection of the only transparent epithelial structures of the body which are exposed to the influences of the air and of the light. In the cornea it is usually the lower half; and of the conjunctiva, preferably that part of the bulbar conjunctiva which is exposed in the eye slit.

The conjunctiva is earlier and more constantly affected than the cornea. Typical is a fatty dryness, which is most pronounced at the thickened wrinkles in the corners, specially during movements of the eye. On examination with the slit lamp, the transparent conjunctiva has become opaque by fine milky droplets, which gradually obscure the underlying vascular pattern. In cases of long standing, a certain amount of hyperpigmentation is always present. The eye white loses its porcelain aspect and looks muddy-greasy, like oil-paint. In such cases Bitot spots are often present, and if they are not, the thick whitish substance of which these are made may be detected as

small slimy tatters somewhere on the surfaces. The Bitot spots develop in the 'Bitot space,' the only place on the eyeball where the white substance can accumulate, that is in the eye slit, at the foot of the slope constituted by the slightly bulging cornea. There they stick to the rough surface, and at these places hyperpigmentation usually becomes most pronounced. The degree of adherence and the consistency of the abnormal secretion vary considerably. The texture is frothy, but sometimes the consistence is like molten tallow. Kreiker (3) supposes that it is whipped into a foam by the movements of the eyelids, but I can hardly accept that these movements are so forceful to result in something like whipped cream. In smears made of the secretion, xerosis bacilli always abound. I wonder if they are not gas-producing in the foam. If we insert a speculum, it is clear that specially the conjunctival folds surrounding the eyeball are thickened and fatty, while the outer parts still are moist. If tear secretion is not too abundant, as in crying, these folds always remain dry. The picture is made up of a certain amount of *1*) redness and injection; *2*) hyperpigmentation; *3*) dryness and opaqueness of the conjunctiva, masked eventually by *4*) a layer of Bitot substance; but the separate contributions of these are variable. The affection is often patchy, but the spot in the Bitot triangle is most constantly affected. As a response to vitamin therapy, the conjunctiva becomes transparent and moist again within a few days, but the Bitot spot, which is not made up by living cells, may remain for weeks, and the hyperpigmentation in the conjunctiva may become more clearly visible.

A small Bitot spot may consist only of a few tiny air bubbles visible in the triangles, specially the outer; a big one sometimes obscures the whole of the eye white by a frothy coating and overflows onto the cornea or into the eyelashes.

The *xerosis epithelialis conjunctivae* (to which I would add, *bulbaris*) is as often as not accompanied by a xerosis epithelialis corneae. This means that the cornea is probably more resistent to the affection than is the conjunctiva. In an average case, the cornea is hazy, rough and dry. The tears do not wet it, or perhaps only for a short interval. It is not sensitive to touch. A few punctate superficial infiltrations or small erosions may be seen on the surface. But the next morning one of these erosions, often in the lower nasal quadrant, may develop into a small dark berry-like protrusion, to which a tiny slip of the iris is attached. From this stage to a hidden or a clear perforation is only one step. The opening rapidly may become larger till part of the iris prolapses, or, as I observed in two cases, the lens is expelled and found in the cot of the child. These weakened patches often are multiple, but still the general shape of the cornea is maintained. The condition is not painful, but the child avoids opening the eyes and is afraid of the light. A certain amount of lacrimation is common. The latter stages are accompanied by inflammatory symptoms, one time resembling conjunctivitis, another time producing a hypopyon and in the worst, but in not rare instances, by a panophthalmia which signifies permanent loss of vision. Now and then the whole cornea becomes paper-like, without patchy epithelial losses, a condition called mummification by Pillat (4–6). Afterwards, the devitalized tissue changes into a huge thick scar, but the eyeball still conserves its shape. The condition of the cornea I consider as xerophthalmia *sensu stricto*, but I am aware that it is called keratomalacia by others.

Keratomalacia, however, is a different process. It is not an ultimate stage of the former, but a colliquative necrosis of the whole of the cornea. It is difficult to describe the first stages but, at least in some instances, it is preceded by a xerosis. Keratomalacia is a rather sudden spongy swelling and melting down of the cornea, with subsequent shrinkage of the eyeball. It is an acute and catastrophic condition, a destruction of the eyeball, eventually without infection, but it is understandable that the adjacent structures become irritated, and that then an eventual xerosis of the bulbar conjunctiva is difficult to demonstrate. It is understandable that the success of vitamin A therapy often is not great, but remaining portions around the lumbus may show a favorable reaction.

The distinction between keratomalacia and xerophthalmia of the cornea is often easy, though not always. Usually kerato-

malacia is symmetrical and both eyes are lost at once, but I know at least of three instances where one eye was affected by keratomalacia and the other by xerosis. I cannot go too far into details, but I believe that keratomalacia is not due to exactly the same etiology as is xerophthalmia. Yap-Kie-Tiong (7, 8) supposes that keratomalacia is more closely dependent on protein malnutrition. If he used the term somewhat loosely I could agree, but he is very keen on the distinction. All cases of serious xerophthalmia belong within the ill-defined limits of protein malnutrition.

The male distribution in keratomalacia is less than in xerophthalmia and the average victim is younger, just as in kwashiorkor; also in the old figures of Mori (9) this is true. In the few serum determinations which we have, the figures for vitamin A and albumin decrease with increasing severity of xerophthalmia. But in kwashiorkor without xerophthalmia both figures are also low. Except morphologically, I do not know if the distinction between keratomalacia and xerophthalmia is so important, but I think that there are suckled infants in a fair state of nutrition who still are suddenly afflicted by keratomalacia (10). As keratomalacia is an emergency condition, it is very difficult to collect reliable data.

If I am going to speak of xerophthalmia in the next minutes, I mean *xerosis conjunctivae et corneae*, eventually with perforations, but I do not include keratomalacia. If I mention keratomalacia, I am considering only the colliquative necrosis.

Several conditions have now been reviewed which traditionally are connected with hypovitaminosis A:

1. Prexerosis, usually combined with hemeralopia
2. Xerosis epithelialis conjunctivae bulbi
3. Bitot spots
4. Xerosis epithelialis corneae
5. Mummification
6. Keratomalacia.

Is the connection always equally close? I am answering the question now from the viewpoint of the doctor in whose environment hypovitaminosis A is common; not of that of the ophthalmologist, who sees all kinds of exceptional conditions. Besides the kind of night-blindness which in a short time responds to vitamin A therapy, I believe that xerosis of the cornea and of the conjunctiva are the most specific symptoms. Bitot spots and keratomalacia may be conditioned by factors which are more complicated. But a Bitot spot is a very useful indicator of the condition. Therefore we cannot bother too much about the few cases of doubtful origin.

There are a few clinical details which I want to consider in passing. Xerophthalmia has been said to be caused by a lack of tear secretion (11, 14). But this is not a symptom common to many cases. Still, it occurs, especially in infants suffering from either advanced marasmus or kwashiorkor, who also may have a diminished secretion of saliva and of sweat. Both may suffer from xerophthalmia as well. I believe that the primary lesion, attributable to lack of circulating vitamin A, is the abnormal keratinization and other degenerative processes in the affected epithelium, as indicated by the papers of Kreiker (3) and of Sweet and K'ang (12). But there are other phenomena occurring in the neighborhood of the eyeball which deserve attention. One is the hyperpigmentation both of the conjunctiva and the cornea. In pigmented people, diverse types of pigment may be present inside the conjunctiva, and there is a lot of variation. But there exists also a fine cloudy pigmentation, which comes and goes in a rather chronic way, according to the nutritional state. It is more closely connected with xerophthalmia than with kwashiorkor, but these conditions often overlap. It may be present before xerosis appears, and it may remain a very long time in the places where the Bitot spots have gone. I doubt if it remains so permanently on a balanced diet, as was suggested by Thompson (13). There is, however, no direct relation to the degree of the affection; rather, more to the duration. If it is a symptom, connected with abnormality of the function of the adrenals, it must be a very early symptom.

A symptom which seldom is mentioned as visible in human patients is enlargement of the pores of the Meibomian glands. They often look like a string of beads along the margin of the eyelids. On inspection of the surroundings of the eye, the skin often looks thin and dry, and fine points of folliculosis may be present. The skin between the eye-

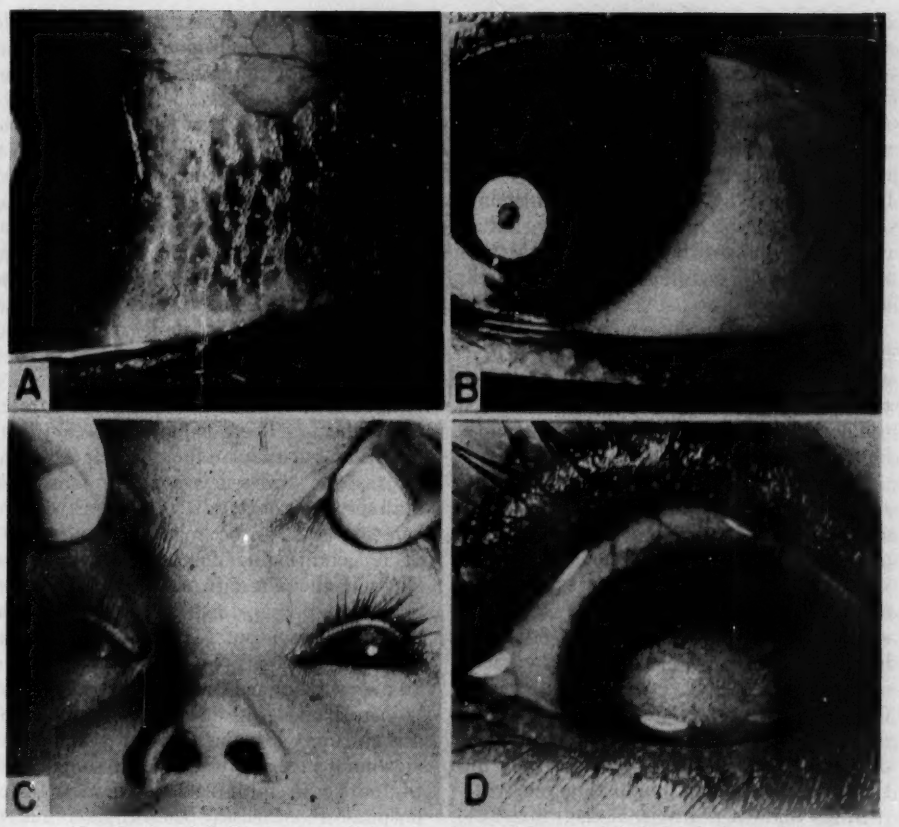


FIG. 2. *A* and *B*, Bitot spots; *C*, keratomalacia with panophthalmia; *D*, keratitis

lashes is usually scaly, but I do not think that the long, dry, broom-like eyelashes which sometimes are conspicuous in malnourished children, are dependent on hypovitaminosis A.

There exists a slight alteration of the conjunctiva in malnutrition, which is characterized by dryness and thinness, lack of vascularization, and with fine wrinkling in the corners. If in such cases hemeralopia is not demonstrable, I doubt the relation with hypovitaminosis A. I do not think that pinguecula and pterygia and such local abnormalities of the conjunctiva present real difficulties in establishing the diagnosis of xerophthalmia. Trachoma is a different question, but as it seldom is frequent before the school age, the differentiation from xerophthalmia is not too difficult.

But too long already we dwell on the symptomatology of hypovitaminosis A, as regards the eye surfaces. Though very convenient and very important from the human viewpoint to observe, these tiny specialized structures of the body certainly do not represent the only manifestations of the lack of such a strategical nutrient. De Gouvea (15) called xerophthalmia, 'ophthalmia cachectica;' Leber (16), 'ophthalmia hepatica;' Bloch (17,) 'dystrophia alipogenetica;' Blegvad (18), 'dystrophia xerophthalmica.' Among the numerous cases, described by Mori (9), the specific symptomatology of kwashiorkor is often easily discernible. In Indonesia, van Stockum (19), de Haas (20) and Oomen (21, 22) stressed the frequency of malignant malnutrition in xerophthalmia patients. Why do xerophthalmia patients die so frequently? (See Blegvad, ref. 18, 25%; de Haas, ref. 20,

33%.) Because, of course, they do not die on account of the eye lesions!

If we do not want to get lost in the labyrinth of casuistry regarding hypovitaminosis A in man, however interesting this may be, we had better concentrate on the environments where the condition is endemic. As in beriberi, sporadic cases of xerophthalmia often have a complicated etiology. Where cases stem more from environmental than from individual factors, the picture usually is less complex.

First we might ask, which other lesions, known from animal experiments, etc., are encountered in patients who are affected by the eye condition? I am speaking now of toddlers, who in my material represent the bulk of the cases.

Are they night-blind, that is, do they suffer from an affection of the retina? The answer is, that probably all are, but in this age group, such is very difficult to demonstrate. That this condition is well known to the mothers is indicated by the fact that a special term is coined in the Indonesian language, and in many of the other languages of Indonesia, signifying 'chicken vision.' Chickens are night-blind. By the way, many of the children are known to stumble at dusk, or not able to enter a spoon right into their mouths, when the light is failing.

Many show a retardation of growth (21). This more particularly regards weight than height. But the significance should not be overstressed, as protein malnutrition has the same effect and the two conditions are difficult to separate. Anyway, we have to conclude that growth retardation is not a typical feature of xerophthalmia and probably of hypovitaminosis A in man.

Some skin affections often are ascribed to hypovitaminosis A. They all refer to abnormal keratinization. I mention xeroderma, mosaic or crackled skin, phrynoderma and folliculosis (23). Do we encounter them very often in the average victim of xerophthalmia, who is a toddler? The answer is 'yes' as to xeroderma, but then, this condition is as common in cases of kwashiorkor or marasmus, without xerophthalmia. A dry, atrophic skin, with demonstrable lack of secretion of sebum or sweat, belongs to the severe cases of malnutrition (24). Again the answer is affirmative as to folliculosis, not so much as to isolated spots over the body, or the extensive spiky conditions which sometimes are illustrated in textbooks, but most frequently seen as a very fine folliculosis in the delicate parts of the skin which surround the eye. Phrynoderma is a problem apart. It sometimes is present in school-age children suffering from less serious manifestations of xerophthalmia, but more often they have no eye symptoms. Only once I saw it in a toddler with serious eye lesions. It then consisted of pointed, horny, pigmented spikes on knees and elbows, more resembling an iguana skin than a toad skin. None of these dyskeratotic conditions easily respond to vitamin A, but on a 'good' diet they gradually subside. There is a connection with nutrition, but they are not striking examples of a single deficiency such as is xerophthalmia. To conclude: some types of dyskeratosis seem to be linked with vitamin A as a conditioning factor, specially those affecting the pores. In my material the best examples, perhaps, are to be found in the large, bulging pores of the Meibomian glands.

A second clinical phenomenon which may be conditioned by vitamin A hypovitaminosis is urinary calculus. From a public health viewpoint it certainly is of much importance to Indonesia (table 2, refs. 25, 26). In my hospital at Macassar the surgeon had to operate on as many cases of urinary lithiasis as I saw frank cases of xerophthalmia. Half of them were young boys, aged about 5–10 years, and most were burdened with *bladder* stones of considerable size; the other half were adults. The boys sometimes showed stunted growth. We learned to make the diagnosis at first view, because of their painful face and their particular posture.

TABLE 2. HOSPITAL PATIENTS WITH BLADDER CALCULI (25): INCIDENCE AS TO AGE, SEX AND INCOME

	Central Java	Batavia	Europe
Period.....	1910–1928	1922–1928	1700–1821
Number....	1471	563	363
0–15 yr.	66%	72	70
16–45 yr.	25	20	30
Over 45 yr.	9	8	

Central Java: 0–6 y.: 40%, 7–15 y.: 26%.
Java: Males, 93–96%; male fraction 0.78.
Batavia: low income group: 88%.

They usually came from far away and from definite areas. A colleague of mine who operated hundreds of cases in Central Celebes contended that they always came from definite valleys. Part of our cases supports his view. Now none of these boys had signs of xerophthalmia and in a few serum determinations we found no abnormal values for vitamin A or carotene. None of my cases of xerophthalmia showed evidence of bladder calculus. A direct relation between hypovitaminosis A and calculus has been denied by several authors (19, 27, 39), and been confirmed by none in Indonesia. The preponderance of the male sex has been attributed to the obstruction to flushing out small concrements caused by the length and the shape of the male urethra. This preponderance, however, hints also at the sex distribution typical for xerophthalmia. Here is a problem that certainly deserves more intensive investigation. All I could say from the clinician's viewpoint is that the bladder calculus of small boys is not strikingly represented in the narrower environment which produces the xerophthalmia patients, but it is there. On the other hand, the environment which provides the young patient with calculus seems to be a typical one in the same sense as that where endemic goiter occurs; furthermore, it is very probable that the previous diet, especially at low age, has been vulnerable. Considering the rather positive evidence, produced by animal experiments, this type of bladder calculus may indicate a conditioned and delayed symptom of hypovitaminosis A (28).

Does the victim of xerophthalmia present other manifestations, which are attributable to vitamin A hypovitaminosis? A high percentage of the xerophthalmia cases suffers from bronchopneumonia and practically none is free from respiratory symptoms. We would expect that bronchiectasis would be of common occurrence. I can neither confirm nor deny this, though I had opportunities for x-ray examinations. The few cases which produced enough evidence sometimes had xerophthalmia, but as often not.

Very few of the patients showed clinical evidence of affection of the epithelial lining of the urinary system.

To know where to look for symptoms in human beings, I have to rely specially on Sweet and K'ang (12), who reported the most extensive series of post mortems. I am struck particularly with the alterations described in glands and the ducts of glands. For instance, enlargement of the parotid glands, which is common in connection with malnutrition, is not a feature belonging either to the acute stage or the recovery period of patients with xerophthalmia. But so far as I know, it is not a condition which is seen at a young age.

In the environment of xerophthalmia patients of Central Java, a rather puzzling affection of the pancreas was discovered by Zuidema (29). It consists of a necrosis and calcification of the gland in young adults from a poor rural background, resulting in cataract and severe diabetes. The disease is wholly different from cystic fibrosis of the pancreas which sometimes produces keratomalacia (30). I will only refer to the condition here, as it possibly may prove to be a conditioned and delayed effect of hypovitaminosis A, too, or may produce secondary signs of hypovitaminosis A.

The principal stock of vitamin A is in the liver. Do we find specific alterations in the liver, which could be related to hypovitaminosis A? Does the loss or the absence of vitamin A in the liver cause any harm to the liver itself? There is very little evidence on this important point and none so far as I am aware from human pathology (31, 32). The limitations of my clinical material do not enable me to give an answer either. But there is one point which I can state. Of about 70 biopsies on xerophthalmia patients, every case showed important alterations of the liver, the most impressive being the fatty infiltration (21, 22; table 3).

This brings us to the point which I tried to avoid up till now. That is, the intermingling of symptoms due to vitamin A deficiency and to general malnutrition respectively; or if you want me to commit myself further, to protein malnutrition. It is evident from the terms which I stated at the commencement of this chapter, that most authors who studied the condition were struck with the general symptoms, that is, the dystrophic state of the victims. Xerophthalmia—speaking now of real cases—is not

TABLE 3. STATE OF LIVER IN 46 CASES OF KWASHIORKOR AND/OR XEROPHTHALMIA. INTERMEDIARY CHANGES OMITTED (21)

Liver	Edema		Xeroph-thalmia		Edema and Xeroph-thalmia	
	Pos.	Neg.	Pos.	Neg.	None	Both
Number (46)	24	22	32	14	4	14
Normal size	1	1	1	1	0	0
Enlarged++	15	9	17	7	2	10
No steatosis	1	1	0	2	1	0
Steatosis++	21	19	29	11	3	13
No fibrosis	4	4	5	3	2	3
Fibrosis++	2	6	6	3	1	0
No infiltration	3	4	3	4	2	1
Infiltration++	1	2	3	0	0	1

a beautiful single symptom, illustrating the effects of a simple deficiency for textbook purposes.

If all that experimenters teach us as regards vitamin A deficiency in animals is true for human beings, then it is quite understandable that we can expect a wide variety of serious general symptoms. Even minor affections of the glands, the respiratory epithelium and the intestinal villi must have a serious impact on the general state of health. We observe lesions and alterations, commonly found in toddlers suffering from kwashiorkor or marasmus, such as: growth retardation, edema, hypoalbuminemia, muscle atrophy and fatty liver. In many cases it is impossible, disregarding the eyes, to say if a toddler is suffering from a vitamin A or a protein deficiency (table 3).

Is it then true that a sufferer from hypovitaminosis A, besides the specific lesions of the eye, always shows rather unspecific signs of general malnutrition? This rather depends on the degree of the eye affection. Many bearers of Bitot spots are normal schoolchildren. Still they may have fatty livers or low albumin levels in the serum. But even in this case, their partners without eye lesions may show similar slight abnormalities (table 4).

Is xerophthalmia an early or a late symptom of hypovitaminosis A? Please remember that this pathological process may be compressed into a few days, resulting in destruction of the cornea, but that it also may be present for six years and not produce more than a hyperpigmentation; further that the course can be intermittent and relapsing. On the whole, I would say that the eye affection is an early symptom, that hemeralopia comes before xerosis, and that conjunctival xerosis comes before affection of the cornea. But as destruction of the cornea sets in, many abnormal keratinization processes throughout the body probably are present too, resulting in a rapid deterioration of metabolic functions, and only one step further, in death. There are no museum pieces of xerophthalmia, showing all the possible lesions, better than those described, on anatomical evidence, by Sweet and K'ang (12) and Wolbach (30). But there often are interfering influences, of a less specific nature, resulting in death before the museum piece is finished.

I would distinguish between chronic or constitutional diseases, which by disturbing storage, transport or resorption, ultimately cause xerophthalmia; and those of a more incidental nature, which act as a pacemaker for the affection. In the former, diet as a causative factor is only of minor importance. Examples of this category are general diseases of the liver, such as cirrhosis, eventually in the wake of a hepatitis, and diseases of the pancreas, such as cystic fibrosis. In my figures on adult patients of xerophthalmia, liver disease, specially cirrhosis and hepatitis, often show up. They lead into a complicated casuistry and deviate from environment and diet, where the real problem is hidden.

The small child, the typical victim, very often suffered from a definite disease a short time before the xerophthalmia lesions became apparent. Quite a number of diverse infectious diseases could be enumerated, where the disease, immediately or after a certain lag of time was followed by an 'outbreak' of serious zerosis. Thus the condition behaved as a *complication* in the elementary clinical sense. In other cases it was known that the child suffered from a simple xerosis before, and that the interfering disease caused an *aggravation*.

Examples of such diseases are: primary phase tuberculosis, primary malaria, pneumonia, measles, chicken pox, whooping

cough and dysentery, either by Shigella or by Entamoebae. Please remember that scarlatina is not a tropical disease, that diphtheria behaves differently too, and that viral hepatitis is not a disease typical for an early age. Then we realize that the common acute infections of childhood often act as a forerunner for xerophthalmia. They may differ as to their nature—protozoal, bacterial or viral—but they all mean an important draft on the body of the child and they are usually accompanied by fever and anorexia. In case of the intestinal infections an interference with resorption is apparent. Then come the slower drafts on vitality, such as—again—tuberculosis, malaria and dysentery. Last but not least are the infestations with helminths, specially Ascaris, but of these I am not so sure, as they are present in everybody. De Haas (20) states "...with these children dystrophy and xerophthalmia go hand in hand, while the vicious circle is closed by the infections mentioned". The imagery, depicting the impact of an infectious agent as the *coup de grace* for a previously weakened subject, is very fitting.

I believe that this conception explains better the actual situation, than the idea of the anti-infection vitamin, which strengthens the tissues and protects against infectious agents. Still the latter concept has some value, as the recurrent attacks, specially at respiratory and intestinal locations may be favored by deteriorations of the epithelium in question.

As already hinted at several times, age is an important aspect in xerophthalmia. Tentatively, I would like to divide the clinical appearance of xerophthalmia in relation to three age periods: *a*) the infant below 1 year; *b*) the toddler between 1 and 5 years; *c*) the school-age child, the young and older adult.

In the first-year child the relative frequency of keratomalacia is high, but cases are rare. Such a child as a rule is breast-fed, and a few are fed on the poor Javanese mothers' or caretakers' ideas of formulas. Before the war, several hundreds had been fed on skimmed sweetened condensed milk out of deceptive cheap tins, which is, according to de Haas (20) an 'ideal' food for providing xerophthalmia in bottle fed infants! Breast-fed or otherwise, the young infant is still influenced by the reserves with which it got under way at birth.

TABLE 4. SERUM LEVELS OF VITAMIN A, ALBUMIN AND GLOBULIN (3, 35, 44)

Group	Vit. A IU %	Alb. gm %	Glob. gm %
Normal			
24 doctors' children	146	4.4	3.2
18 schoolboys, rich	100	4.2	3.3
38 schoolboys, poor	68	4.0	3.7
28 orphanage	77	3.9	3.4
21 poor area	44	3.5	3.5
Patients			
11 xerosis conf.	33	3.5	3.4
11 xerophthalmia I	3?	2.7	3.5
10 xerophthalmia II	9	3.0	3.2
10 keratomalacia	7	1.7	2.6

If a newborn infant has already keratomalacia at birth, the probability is great that no such reserves existed. If the child was a twin or a premature, we have a ready reason to suppose that something was wrong with the reserves. If a mother of a suckling with xerophthalmia is proved to suffer from cirrhosis of the liver—and perhaps is night-blind herself—then she probably poisoned her child with her deficient milk. If a young infant has fever and a parasitemia while presenting xerophthalmia, then the malaria attack was the last straw. But if the mother was in good condition, her milk satisfactory and her infant well, we do not understand the mechanism (10, 34). I am quoting now a few instances, of which I know, but as these very young cases are rare and often do not come under observation, I have to stop here. It will be clear that further research into the causation of xerophthalmia and keratomalacia in breast-fed infants will show what part is played by intrauterine and mammary feeding, respectively.

Now comes the second year, perhaps the worst one in the nutritional history of the Indonesian child (35). A certain percentage is already weaned, another gets only a little—say 100 cc—breast milk, and has no other sources of vitamin A. But the second year is the typical age for kwashiorkor, not for xerophthalmia. Xerophthalmia creeps through the whole toddler period in a rather uniform shape and serious corneal xerosis forms the backbone of all cases. In addition, as we will see later, there is a definite attack rate as to sex. The toddler then is less bound

TABLE 5. MALE DISTRIBUTION IN AGE GROUPS OF CASES OF XEROPHTHALMIA FROM CENTRAL JAVA (41)

Age, yr.	Jogjakarta			Semarang		
	Total cases	Male		Total cases	Male	
		Number	%		Number	%
1	267	155	59	52	31	81
2	781	446	56	167	98	59
3	1708	1019	59	348	201	58
4	1513	872	58	223	126	56
5	713	419	59	66	40	61
6	324	210	65	31	18	58
7	159	105	66	12	9	75
8	93	68	73			
9	86	59	69			
10	32	25	78			
11	36	31	86			
12	23	21	91			
13	47	40	85			
14	36	31	86			
15	30	29	97			
16–24	203	174	86			
Over 25	240	207	86			

to the body of the mother and more exposed to the promiscuity of his surroundings and worms, bacteria and viruses find him an easy and unprotected prey.

From school age on the picture alters. The child is more independent as to diet. The stress of a rapid growth rate, except before puberty, is smaller. The body already acquired a certain resistance through infections. A fatty liver still often is present but the degree is less. The number of xerophthalmia cases rapidly dwindles, the serious cases are exceptions, and simple xerosis and Bitot spots are far in excess. This goes on through puberty, which does not seem to make much difference, and later on through pregnancies and lactation periods, which, curiously enough, do not so very much favor xerophthalmia as the statistics on hemeralopia would suggest! It is the male, again, who suffers most, and the male fraction moves to a higher level. Probably many cases then suffer too little to bother with doctors or nurses and the adult with slight symptoms is probably too less represented in my material. But the real victims still include instances of keratomalacia, and among them the number of sufferers from chronic liver disease is great. I would very much like to say more about xerophthalmia in adults, specially females, but I cannot as the latter are extremely rare. I could, however, refer to the papers of Pillat (4–6), whose Chinese patients were mostly adults, but males.

In Indonesia, before the war, an argument was going on whether xerophthalmia favored the male sex and whether it considerably influenced the number of the male blind (36, 37, 40, 41). Tijssen (38, 40) contended that keratomalacia was a deficiency disease of the first year of life, of which males and females suffered in the same proportions. He regarded xerophthalmia as a constitutional disease of older children which only affected predisposed persons, of which he stated that there were 2 boys to 1 girl (that is, a male fraction of 0.67). Not only Tijssen's conception deserves attention, but also the way on which he came to his conclusions. He was a traveling ophthalmologist in north Sumatra, and his figures stem from careful field observations.

That is more than I can say from some of my statistics which, though extensive, are derived from hospital entries. They show a definite distribution between the male and female sex, which appears in every environment, where figures were collected in a similar way (41). While making provisionally a mental reservation for keratomalacia and for the xerophthalmia cases which occur in the first year, I came to believe that there exist definite attack rates for the sexes, and that these change with increasing age. The male fraction of xerophthalmia cases between 1 and 5 years is about 0.58, while above 10 years it is about 0.85 (table 5). The figures in question come from city hospitals in three different places, but more than half of the cases are from rural surroundings. The numbers of case histories scanned are 11,000, 1000 and 160, respectively. These specific male fractions do recur so stubbornly that I cannot accept that they they are biased by extraneous influences, as pretended by some authors (42, 43). So far discernible, they also do occur in the distribution of cases outside Indonesia, to begin with, in the 1470 cases described by Mori in 1904 (table 6).

With the reservation made above, as for me the argument of the sex distribution in xerophthalmia is closed, though minor points still require investigation. But I am happy

that it is not my task to go into the pathogenesis of the phenomenon. Even if I am not so sure about the next points, I believe that the age distribution in the sexes is the same. That is, that the affection does not come earlier or later in males than in females (table 5). Also, I could make no difference between the degree of xerophthalmia in males and in females.

I hope that by now you will not shrug your shoulders and tell me: "We have known for a long time that the male sex is more vulnerable than the female." Kipling also stated that the female of the species is more deadly than the male. But the question is, has this something to do with vitamin A? If we look at the male fractions of stillborn, of first year deaths, of congenitally blind, of young blind for other diagnoses than xerophthalmia, of young blind either in neglected as well as in well-nursed countries, we find figures that at least rhyme curiously with the sex distribution for xerophthalmia, (table 7). I will not stress further this point, as it is not so much interesting from an epidemiological than from an etiological viewpoint.

I will now try to answer the question, who is suffering from xerophthalmia in Indonesia and why. As you are aware, I am considering xerophthalmia as an endemic disease in that country, and therefore I will concentrate on the relation between environment and the victim.

First—how many persons show evidence of xerophthalmia? This is a tricky question, as much depends on the definition. If prexerosis is excluded, and xerosis and Bitot spots are considered as clear indications of the condition, I myself have only one figure on the incidence in a random sample of the population (41). Once I discovered 11 cases among 415 children from 1 to 5 years old in a typical quarter of Djakarta. The general incidence will be between 1 and 7% in this particular group (North Sumatra, ref. 39; Central Java, ref. 17). That is enough, by the way, to be responsible for a considerable part of toddler mortality and adult blindness! Serious cases do not live long! Again, xerophthalmia is a disease particular to the toddler period (table 8). To conceive an idea of the potential danger this group specially should be examined about the prevalence of the condition. Though more convenient to handle, school children cannot furnish the same type of information. Among the latter an incidence of Bitot spots of 1–5% was several times reported.

TABLE 6. MALE FRACTIONS IN GROUPS OF DIFFERENT ORIGIN

Origin	Age Group	Male Fraction
Indonesia (S)	1–5–	0.58
Indonesia (J)	1–5–	0.58
Indonesia (M)	1–5–	0.60
Japan 1904*	1–14	0.57
Belgian Congo†	mixed	0.84
Indonesia (J)	10+	0.86

S = Semarang, J = Jogjakarta, M = Macassar.
* Mori (9);† Roels, *et al.* (51).

In the eye hospital of Dr. Yap-Kie-Tiong at Jogjakarta I studied 11,000 case histories from 1934 to 1954. There a separate register is kept for the private practice. After scanning some thousands of entries and not encountering one case of xerophthalmia, I gave up and decided that they do not occur in the more prosperous part of the local population. Mori (9) reported that after raising the consultation fee, the numbers of observed cases decreased considerably! Xerophthalmia is a disease of the low income categories in urban, and of the low standard categories in rural, surroundings. Vitamin A deficiency is a condition "qui s'arrête devant le galon" (45). But if in rural regions the young child does not get what the table of the adults can provide, cases of malnutrition or xerophthalmia may develop, even in not so bad off families.

Naturally there are differences between the diverse regions. In Central Java the endemicity is apparently striking, but then much attention has been paid there to the condition. In the South Celebes where the population density is also high, the affection rate certainly is much less though I encountered 50–80 cases yearly in one of the big hospitals of the region. Altitude does not much matter. In Bandung, West Java, and in North Celebes at 3000 feet many cases are present. Gomperts (39) reported an incidence of 8% in the high valleys and of 5% in the coastal plains of Atchin. The vicinity of the coast of the big rivers, like on Borneo, implying the possibility

TABLE 7. MALE FRACTIONS IN DIVERSE POPULATION GROUPS (47)

Origin	Period	Condition	Male Fraction
Indonesia (J)*	1953	live births	0.509
Indonesia (J)	1953	stillborn	0.58
Indonesia (J)	1953	first year deaths	0.55
Indonesia (WJ)	1930	young blind, 2–15 yr.	0.57
Indonesia (NS)	1930	young blind, 2–15 yr.	0.60
Netherlands	1920	young blind, 3–15 yr.	0.58
Netherlands	1920	congenitally blind	0.67
England	'91–'38	first year deaths	0.56
England	'51–'54	blind, 0–1 yr. all diagnoses	0.56
England	'51–'54	young blind, 5–15 yr.	0.58
U.S.A.	1900	young blind, whites, 5–10 yr.	0.58
U.S.A.	1900	young blind, colored, 5–10 yr.	0.59

J = Jogjakarta, WJ = West Java, NS = North Sumatra.

of fish consumption, are quite compatible with the presence of xerophthalmia (46).

Only on the island of Flores cases seemed to be rare, though the nutritional state of the toddlers was anything but satisfactory. Now the Florinese are yellow maize eaters and in addition they consume good quantities of green vegetables. In fact, I discovered several cases of xanthosis in small children, probably to be ascribed to hypercarotinemia. Though the protein portion of the diet is low, the quantities of carotene consumed are many times the average of a Javanese child, also in schoolboys of a certain region (Gunung Kidul, table 9, ref. 44).

Another factor affecting the frequency is season. In some regions cases come up with the flies and the dysenteries towards the end of the rainy season; in others a wave of xerophthalmia follows a wave of measles. The influence of climate on the large material of Jogja was not very striking. This city is situated on the flanks of a big volcano and there are many contrasts in rainfall in the immediate surroundings during the year.

The influence which, to all appearances, dominates most the frequency of xerophthalmia in Indonesia is age. There is a low percentage (2–8%) in the first year, followed by the bulk of the cases in the 2nd–6th year (table 5). What remains after that is only a trickle of ½ per cent or less per year through puberty and adult life. All our figures for separate groups, either regional or for the sexes, show a peak in the third and fourth year, that is at least one year after the peak incidence for protein malnutrition. How is this peculiar distribution connected with the provision of the organism with vitamin A?

A large percentage of the Javanese mothers rarely have access to animal foods. Thus their only source of vitamin A is carotene. The figures on blood levels, collected by van Veen and Lanzing (42) do not show big differences between the percentages for males and females. In rural surroundings 72–98% of the adults examined had over 50 IU per 100 cc (table 9). The average level for Djakarta mothers as indicated by de Haas (47) is 60 IU. This is lower than averages elsewhere, but most of the mothers do succeed apparently in giving enough vitamin A to their child via the breast. In a few mothers of sucklings with xerophthalmia the breast milk did not contain any vitamin A. It is yet unknown how much vitamin A breast milk should contain, to avoid xerophthalmia in the child, or which are the causes of a low level in the milk. An incidental discovery of liver cirrhosis in the mother of one of our patients makes us think. A systematic investigation on the relation between carotene intake, and blood and milk levels in Indonesian mothers seems very desirable. The etiology of xerophthalmia in breastfed infants cannot be understood in another way.

If the small baby is deprived from the breast and artificially fed on deficient food, the causation becomes understandable. Such cases of xerophthalmia do regularly occur. Particularly tragic was the frequent occurrence of xerophthalmia, which I mentioned, in Djakarta infants fed on sweetened condensed skimmed milk before the war (20). Though this dangerous product was driven from the market with much trouble, skimmed milk powder reappeared in latter years. The importer now was UNICEF! But nowadays the implications are better understood.

I am not satisfied on the answer I had to give on the not too rare occurrence of xerophthalmia in breast-fed sucklings. The question why it occurs so frequently in toddlers is still more complex. Most puzzling is however the problem, why so many poorly fed toddlers do *not* develop xerophthalmia.

Popular first foods in Indonesia are cooked rice and ripe bananas (seldom of the yellow varieties). They provide very little carotene. The amount of yellow fruits, yellow sweet potatoes and green vegetables, which the child gets in the second year are very limited. In many cases we have to assume that as to vitamin A metabolism, the child has to economize, either on its prenatal reserves, or on the trifle which it still gets from the breast. In contradistinction with our findings in Papuan mothers (48), I believe that most Indonesian mothers have very limited breast milk in the second year. Studying the diets of Djakarta toddlers, I found that both protein and carotene sources were scarcest in the second year, while towards the fourth year only the intake better corresponded with the opportunities of the adult diet (35). In the country, the same thing may happen, but sometimes the quantity of green vegetables consumed is anything but small. Tijssen (38) insisted that even the latter children may become a victim to xerophthalmia if they have the predisposing constitution. I myself can cite several examples from Celebes as well as from Central Java, that children apparently consume a fair quantity of carotene and still present xerophthalmia (35). I have to conclude that not only the absence of carotene in the diet, but also the 'apraxia' of carotene, are to be held responsible. That carotene does not produce the theoretical equivalent of vitamin A under many circumstances, has often been proved, experimentally and clinically, since the acute deductions of Tijssen. In India, Aykroyd and Krishnan (49) made observations of the same bearing. But I have no opinion to voice on the relation between genes or constitution and xerophthalmia, though. McLaren's paper (50) made me think of racial differences.

The fact that carotene has not the same activity in the child as it has in the food composition tables is of serious consequence.

TABLE 8. PERCENTAGE DISTRIBUTION OF XEROPHTHALMIA (AGE 0–10 YEARS)

Locality, Year	Infant		Toddler					Schoolchild
	No.	I	II	III	IV	V	VI	VII-X
Jogjakarta, 1954	1301	8%	12	30	26	12	5	4
Semarang, 1954	890	6	18	38	24	8	4	2
Macassar, 1957*	166	2	11	24	24	21	11	7
Japan ± 1900†	1475	2	9	18	28	20	10	13
Denmark ± 1920‡	429	85	8	3	1	1	0–1	3

Differences, in relation to incidence as to age, of xerophthalmia in various endemic. In Japan formerly, the predilected victim was the toddler as it is in Indonesia nowadays. In the classical endemy of Denmark it was the infant below 1 year.
* van Wisselingh (115); † Mori (9), 1904; ‡ Blegvad (18), 1924.

Many of the toddlers with xerophthalmia suffer from intestinal disease, and practically all have an affection of the liver, even if fatty infiltration can perhaps not always be called a lesion. They have a diet which is poor in protein and fat. Their general state much resembles the condition of children with protein malnutrition. All these factors eventually conspire against the resorption and conversion of carotene and, possibly, the storage of vitamin A.

In fact, it may be not so much the low intake of carotene which ultimately is responsible for the toddler-xerophthalmia, as well as the secondary influence exerted by other limitations of the diet on vital tissues. The close relations between protein malnutrition and xerophthalmia strongly favor this suspicion. So far available, the figures on the dependence of serum levels on dietary intake, do not favor a simple explanation (table 10). In passing, I may refer to the studies of Roels (51) in the Belgian Congo, which strongly imply that the minor degrees of xerophthalmia observed there are conditioned by the low fat intake.

Another factor which much influences the incidence of xerophthalmia in the toddler period is the frequency of infectious diseases. The age distribution of the common diseases of childhood, including the intestinal, would probably very much coincide with the increase and decline of the curve for xerophthalmia. There are, however, limitations. The affection rate for malaria attacks probably would come earlier, and the rate for primary phase tuberculosis perhaps later. To conclude, important agents, which I advocate to explain the relatively high frequency of

TABLE 9. SERUM LEVELS OF VITAMIN A UNDER 50 IU (42)

	Child <15 yr.	Adult
Tjugenang	59%	2
Tjikundul	73	13
Notopuro	69	28
Gunung Kidul	69	18

v. Veen & Lanzing (42), 1940, few children under 5 yr.

xerophthalmia in toddlers in Indonesia are (tables 11 and 12):

1) The malnutritive tendencies of the diet in the toddler period, not only as regards the limitations of carotene intake, but especially the depression of carotene activity by the direct and indirect consequences of a low protein, low fat diet.

2) The frequency of many contagious childhood diseases special for this period. The variety of the diseases preceding the outbreak of xerophthalmia seems to indicate that it does not matter very much whether this is a protozoal, bacterial, viral, respiratory or intestinal disease. There may be, however, additional influences!

I realize with some mortification that if I succeeded in making the epidemiological picture a little clearer, I got no further than a circumlocution of de Haas' statement: "With these children dystrophy and xerophthalmia go hand in hand, while the vicious circle is closed by infections" (47). Some consequences portentous for preventive measures, however, came to the fore. Do not rely on carotene if it is humanly possible to provide the vitamin itself! If you have to rely on carotene, make sure that it works by creating the necessary conditions, that is: enough protein in the diet, and possibly some extra fat! As I, for several reasons, do not believe in non-dietary measures to prevent xerophthalmia in the long run, these are the only principles to which I confess for eradicating this horrible disease.

Up till now I tried to tell you all I know about hypovitaminosis A, in accordance with the invitation to this conference. Follis did not ask me to dwell on how I feel about xerophthalmia. Do please allow me to tell this, too.

Xerophthalmia has been the most bitter pill for me to swallow during 18 years of doctor's work in Indonesia. The over and over repeated experience of discovering a child, recently blinded, in the arms of the mother; having to tell her that I now could do nothing more to save its eyesight; remembering that I could have done so with a few spoonfuls of cod-liver oil some days ago; these things still enter my nightmares. They belong to the most vivid examples of what disprivileged people in underdeveloped regions sometimes miss.

Now what do I find in textbooks and scientific publications if I want to have real information about this horrible, though easily curable condition? Textbooks on tropical diseases tell you everything about Rift Valley fever, melioidosis and blastomycosis, but they sometimes do not spend one word on xerophthalmia. Every nutrition journal flows over with vitamin A, for instance, in heifers, turkeys and ducks which are eaten by the best-fed people of the world. More printing space nowadays is devoted to a few cases of hypervitaminosis A, induced by an irresponsible vitamin racket, than to the thousands of small children who die or get blind every year due to the lack of a handful of vitamin A units. What on earth is nutritional science good for, if, even in the atom age, it is not capable to counteract one of the foulest consequences of bad nutrition? Do you realize, that since the days of Mori, 60 years ago, not in Japan, but in countries like Indonesia, not one step forward has been taken, in spite of a mountain of thoughtful attention paid by doctors?

By thus exposing my feelings, I hope that the organizers of this conference understand how grateful I am for this opportunity, and how obliged, gentlemen, I am for your attention.

RAMALINGASWAMI: Oomen made a distinction between a mummified lesion of the cornea, as representing the end results of xerophthalmia, and keratomalacia. He implied that keratomalacia may have no relationship to vitamin A status, and related it to protein deficiency. Our experience, both with clinical and experimental vitamin deficiency, prompts us to think that perhaps this kind of differentiation is not justified. Both seem to be very similar pathological processes, and the problem is related to the acuteness of the development of vitamin A deficiency rather than any fundamental

difference in the etiology of the two. We have observed children with keratomalacia with a history of short duration, including an acute diarrhea. They did not have Bitot spots, but many of them showed a soap-bubble appearance of the conjunctiva and a generalized affection of the cornea. We feel there is really no fundamental difference and that the response to vitamin A in both conditions is excellent. I think Oomen has raised a fundamental problem. I would like to ask him further, in view of the very extreme changes of vitamin A deficiency which he finds in the children, whether he has any experience concerning what changes are found in the nervous system, and whether there is any evidence of congenital malformation, with special reference to congenital hydrocephalus. He made a point about urinary calculi. We have been studying this problem for some time. It appears that the children whom we see in Delhi, with bladder stones, who are more or less of the same 5- to 10-year age group as Oomen mentioned, do not show obvious clinical evidences of vitamin A deficiency. We are studying them from a metabolic point of view. I doubt very much whether one can relate calculi primarily to a vitamin A deficiency.

OOMEN: I thought that I had made it clear that if there are distinctions between keratomalacia and xerophthalmia, there are also very close links. I mentioned, sir, three examples which I have seen myself, of one eye being affected by patchy xerosis, and another, which I have illustrated elsewhere (21), of one eye being affected by mummification and the other by keratomalacia, in the same child. I think that this proves enough that the conditions are very much affiliated; but on the other hand, keratomalacia seems to have a slightly different epidemiology from xerophthalmia. I mentioned, for instance, the age and, possibly, the sex distribution. It is not always easy to make the distinction, especially if one is studying the case histories of other doctors. This is not only an observation or a deduction of mine, but if you look at the figures of Mori (9), you will find the same thing. So please do not imagine that I think keratomalacia has nothing to do with vitamin A; but I still think it is useful to make a distinction between the colliquative necrosis affecting the whole of the cornea and the patchy epithelial defects in xerosis of the cornea.

TABLE 10. DIET AND SERUM LEVELS (AVERAGE CHILDREN 5–15 YR.) (44)

	Doctors'	Orphanage	Poor Area	Males
	Diet			
Calories	2210	1470	1270	1010
Protein	70	37	29	10
Animal prot. %	33	1	2	0
Vit. A, potency	2600	4050	800	3360
Vit. A, real, %	23	1	0	0
	Serum			
Albumin g 1%	4.3	3.8	3.5	3.2
Alb/glob.	1.4	1.1	1	0.9
Carotene IU 1%	150	140	70	150
Xerophtol IU 1%	160	80	40	120

Now about congenital deformities. That is naturally more difficult to answer. You will understand that I thought about it as soon as I saw publications on rabbits representing hydrocephalus. I have seen cases of hydrocephalus come into my hospital every year, say 3 or 5. But I have never thought that there was a connection with vitamin A; rather, a connection with genetic factors. I am afraid that I cannot go further than stating this, because it was beyond my horizon as a hospital doctor.

AYKROYD: I never observed nervous changes, in the sense of beriberi, in children affected by xerophthalmia. I have not enough evidence on adults to make a definite statement, but my impression is that no typical neurological symptoms, peripheral or central, were connected with xerophthalmia in children or adults.

MCLAREN: It has seemed to me that the problem of the possible role of protein deficiency in the etiology of keratomalacia as seen in clinical material is too complicated by trauma and infection to yield satisfying answers. I have studied this somewhat in the rat, and I shall very briefly mention later work which indicates that low protein diet accompanying vitamin A deficiency does not enhance the signs of xerophthalmia, but they are put off considerably. Further, I have studied the effect of injuring the cornea by a thermal burn in animals on diets in which vitamin A was deficient on the one hand, and in which vitamin A and protein were deficient, on the other. In various com-

TABLE 11. ETIOLOGY OF XEROPHTHALMIA (DIETARY LIMITATIONS)

a) Vitamin A insufficient Diet otherwise complete (primary deficiency)	infants, Denmark, 910–1920 (18) infants on skimmed milk, Djakarta 1935–1940 (20) certain breastfed infants
b) Vitamin A insufficient Carotene satisfactory Low protein diet (conditioned deficiency)	toddlers, Japan, 1890–1900 (9) toddlers, Indonesia (21) camp conditions, India (49)
c) Carotene satisfactory Low fat diet	schoolage children, Ruanda-Urundi 1956 (51)
d) Diet temporarily insufficient Poor reserves (seasonal deficiency)	breastfed Russian infants after Lent 1910–1920 (116) seasonal increases, Indonesia
e) Low vitamin A, low tocopherol	hypothetical

binations of these deficiency factors, I have not been able to demonstrate any delay in the healing of corneal wounds. I don't think we should overemphasize this point. I have talked with Yap-Kie-Tibing personally about it, and I am not impressed by the results that he has published on 10 cases of xerophthalmia, and 10 cases of what he calls keratomalacia, though he does not give a clinical definition. There are a number of objections to his work. A striking one is that he told me that his keratomalacia cases usually did not survive more than 12 or 24 hours after admission. I think there is a good deal of evidence, both published and unpublished, in his work, that protein deficiency is a paramount factor in all this. I'd just like to comment on Oomen's sex difference in xerophthalmia. The number of male children who are admitted might be more than the female; moreover, were other factors controlled? The number of cases coming to a hospital will not give us any idea of the incidence of the condition in the population. I think it's very risky to draw conclusions from that. Apart from these facts, I would wonder if there is statistical significance in the differences Oomen observed.

OOMEN: I cannot go further in distinguishing between protein malnutrition and vitamin A deficiency than stating that the same environment which produces kwashiorkor cases in the 2nd year produces xerophthalmia cases, more or less combined with protein malnutrition, in the 3rd or 4th year. I have not been in a position to make a distinction in the background of the two conditions, but I think that I can state at least that in these patients protein malnutrition and xerophthalmia are very closely linked together.

Now about the sex question. The significance of the differences is dealt with in my published tables (41). The objections raised by McLaren have been made to me several times. Some people suggested that parents bring more male children to the hospital than females. I don't think that it is true, because on the whole the numbers of male and female admissions were about equal. I cannot think of any reason why they should prefer the male above the female. The patients were not Chinese, but Javanese. Some people told me that girls have better access to the family pot or to the kitchen than boys have; or that boys consume less carotene for prestige reasons. I don't think that there is truth in this, because I am speaking of small children, 2, 3, or 4 years old. I think that such factors cannot be responsible for a big difference. Then again, there is a suggestion that boys were more exposed to sunlight and sports than girls were. If you look at the behavior of small Javanese children, I don't think there are differences which could explain that. The only thing one can state is that boys are just a little heavier and just a little taller, by half a kilo and by a few centimeters, than girls are. If my statistics are biased, I think they would be biased in the higher age groups, and more related to age than to sex distribution.

HIGGINSON: I would just like to remark that there are areas where phrynoderma is widely prevalent and xerophthalmia is very rare. Secondly, I would like to ask whether there are actually skin lesions in humans that are attributable to pure vitamin A deficiency, and thus curable? From what I've heard, there seems to be some doubt on this point.

SCRIMSHAW: May I speak only to the first point, the relation of protein malnutrition to skin lesions in vitamin A deficiency. Cases of kwashiorkor can be very, very severe in Central America, with skin lesions covering the entire body, and can be of long standing, without eye lesions reaching this degree of severity. At the same time, in kwashiorkor there is consistently a considerable degree of

vitamin A deficiency, as evidenced both by lowered serum levels and by very low contents of vitamin A in the liver. However, these seem to be only slightly lower than in the population from which the cases come. There is another mechanism beside dietary deficiency operating to affect vitamin A metabolism in kwashiorkor. When vitamin A is given to cases of acute kwashiorkor in a single oral test dose of 75,000 μg, with a test meal given 2 hours after the initial dose, it does not appear in the blood stream (fig. 1). However, after 5 days, and sometimes after 4 days, of optimum therapy, the same dose of vitamin A results in a very substantial rise in serum vitamin A. Nevertheless, despite the influence in the Central American cases of relatively deficient diets in vitamin A, a reduced absorption of vitamin A, and probably a reduced ability to convert carotene to vitamin A, severe eye lesions do not occur.

TEJADA: I have a question to ask Oomen; as far as Central America is concerned, the prevalence of renal and bladder stones in children is very seldom observed. I have had the opportunity of seeing some children in Guatemala with fibrosis and calcification of the pancreas with definite signs of vitamin A deficiency. I wonder if these signs are not secondary to the pancreatic dysfunction. What is the incidence of fibrosis and calcification of the pancreas Oomen has observed in Indonesia?

PATWARDHAN: Oomen has pointed out the lack of association between xerophthalmia with xeroderma. In the earlier observations on the ocular manifestations of avitaminosis A, mention was often made of this association with phrynoderma. Oomen's observations, as well as a large number made in India, show that the peak incidence of xerophthalmia and phrynoderma occurs at different ages. Aykroyd and Krishnan, as far back as 1935 (49) showed that in Malabar, a part of India where the incidence of xerophthalmia was fairly high, children had a smooth skin, showing that xeroderma and xerophthalmia do not always go together. One might ask, if such is so, how could they both be due to vitamin A deficiency? The answer is to be found in therapeutic tests and in certain biochemical investigations. Gopalan showed in 1946 (53) that phrynoderma could be cured by administration of linseed oil when administered with the vitamin B complex. He felt at the time that vitamin B complex had a role to play in its etiology. Later on, Menon, Trilpule and Patwardhan (54) studied about 50 cases of phrynoderma, in which treatment with sesame oil containing essential fatty acids was compared with treatment with vitamin A. These authors demonstrated that administration of a fat which is rich in linoleic acid brought about a marked improvement in phrynoderma. We also determined the iodine value of the serum lipids, and found that clinical improvement was accompanied with an increase in iodine number, which was depressed in untreated cases. To my mind, therefore, it appears that these therapeutic trials, together with the biochemical evidence referred to above, are a clear indication that phrynoderma and xeroderma are not due to deficiency of vitamin A in these cases. One finds xeroderma as one of the commonest signs of malnutrition in poor-class children in the tropics. This responds to vegetable oils containing linoleic acid. Scrimshaw's observations that phrynoderma did not respond to treatment with vitamin A for about 1 year would support my contention that vitamin A deficiency is not the primary causative factor.

I would like to ask two other questions.

TABLE 12. ETIOLOGY OF XEROPHTHALMIA (INDIVIDUAL LIMITATIONS)

a) Resorption vit. A impaired	defective carotene resorption, many pathological conditions and in infants
Exogenous influences	paraffin, atebrin, *helminths*
Endogenous influences	pancreatic disease
	absence of bile
	intestinal disease
	fever
b) Carotene conversion impaired	*hepatic and intestinal disease*
	phosphor intoxication
	atebrin
	infants
c) Blood transportation blocked	*fever*
	acute hepatitis
d) Liver storage disturbed	*measles*
	hepatic disease
e) Increased requirement	prematures
	pregnancy
	males
f) Reduced intake	*custom*
	anorexia
	many digestive and general diseases

Scrimshaw indicated that for 4 days children suffering from kwashiorkor have defective absorption of orally administered large doses of vitamin A. We know that sometimes keratomalacia is associated with kwashiorkor and that its progress is halted by oral administration of large doses of vitamin A. If Scrimshaw's observation is correct, how can one explain the above effect?

My final observation refers to the remark concerning corneal pigmentation, a type of pigmentation which has been seen by us in India, and which was originally ascribed to a deficiency of vitamin A. The pigmentation is 'round the cornea but separated from it by a thin layer of clear conjunctiva. As you will see in this picture in my monograph (55), it may be a full circle about the cornea, a semicircle, or it may be in broken segments, but separated from the cornea. You cannot mistake it for the arcus senilis. The degree of pigmentation varies. We have studied vitamin A levels in the blood, and determined the impairment in dark adaptation, and have finally administered vitamin A for prolonged periods. We have found that this particular type of pigmentation, which may also be accompanied by patchy pigmentations of the bulbar conjunctiva, is not a manifestation of vitamin A deficiency.

IV. The Pathology of Vitamin A Deficiency

Mason: The experimental approach to vitamin A deficiency really began with the early studies of Goldschmidt (56), and Mori (11), who described the cornification of the conjunctiva in the rat, and similar changes in the lining of the pharynx, trachea and ducts of salivary glands. Wolbach and Howe (57, 58, 59) later extended the picture and emphasized the wide distribution of these effects on lining epithelia of the body, both in the rat and guinea pig, calling attention to the fact that epithelia of salivary gland ducts were usually first affected, then those of respiratory and genitourinary passages, and later those of the periocular gland ducts and conjunctiva. Skin changes, which became apparent later, have been described as atrophic rather than hyperkeratotic, such as had been described in avitaminosis A in man.

The important thing to keep in mind in regard to the epithelial changes in experimental animals is that the specialized functions are suppressed and there is gradual transformation to a stratified, keratinized type of epithelium. This is not a permanent effect, as indicated by the ability of such epithelia to recover and reconstitute themselves, following vitamin A therapy, whether they be cuboidal or columnar epithelia of gland ducts, stratified squamous type as in the cornea, transitional type as in the renal pelvis and the bladder, or more highly specialized pseudo-stratified columnar, ciliated, mucus-secreting type as in the respiratory tract. It is indeed remarkable that such a highly differentiated epithelium can be so drastically altered by vitamin A deprivation and yet retain the capacity to be reconstituted following vitamin A therapy, provided intercurrent infection does not occur. The term 'keratinizing metaplasia' of epithelia in vitamin A deficiency, as applied by Wolbach, is a very appropriate term.

At about this same period there appeared the pioneer studies of Frazier and Hu (60), Nicholls (61), Loewenthal (62) and others, on follicular hyperkeratosis and phrynoderma in vitamin A deficiency in man. The significance of these lesions has been a subject of debate. There are suggestions that they may represent effects of a combined deficiency of fatty acids and the vitamin. There appeared also in reports by Blackfan and Wolbach (30) and Sweet and K'ang (12) the first extensive postmortem studies representing 28 cases of vitamin A deficiency (25 children under 4 years of age and 3 young adults). The same widespread keratinizing metaplasia noted in experimental deficiency was observed. It is surprising indeed that no significant pathological observations in man have been added since that time.

Efforts to reproduce ocular changes in the monkey have not been very successful. Hetler (63) pictures typical xerophthalmia with keratomalacia in one monkey, and describes extensive keratinization of other epithelial tissues in her experimental animals.

During the 10-year period of 1933 to

1943, experimental studies were concerned extensively with neurological lesions and bone changes first observed in pigs, and later in cattle and other species. There were many controversies as to whether the papilledema, blindness, motor incoordination and spasms observed were due primarily to nerve lesions, demonstrable in peripheral and central nervous pathways and nerve cells, or secondarily to bony changes causing constriction of the optic nerves and other portions of the nervous system. Some, particularly Mellanby (64), attempted to relate xerophthalmia and other epithelial changes to loss of neurotrophic control, following degeneration of peripheral nerves. The studies extended to the rat and dog. It soon became apparent that these two phenomena were unrelated. Then came the extensive studies of Moore (65, 66) on cattle, where constriction of the optic nerve by overgrowth of bone seemed to explain most of the phenomena observed. Mellanby (67) also concluded that the deafness which he observed in dogs could be explained on the basis of an imbalance in bone growth. Wolbach and Bessey (68) clearly showed that in the rat the nerve lesions were the result of disproportionate growth between bone and nervous tissue, the primary change being in the skeleton, and affecting particularly the epiphyseal cartilages, retarding their development but not affecting appositional bone growth, resulting in a thickened but shortened type of bone. At the present time, most investigators are agreed that, in mammalian species, the nervous system changes are secondary to bone changes.

We might ask ourselves now, "What have we really learned from these studies?" The nerve lesions and symptoms seem to be secondary to some retardation and disorganization of bone growth, either locally or generally, affecting particularly the brain and cord. How can we correlate these with the epithelial lesions? Is there any primary metabolic disturbance which might relate to both? There are suggestions only, based on a limited amount of evidence, that this might possibly relate to some effect on the production of either the quantity or quality of mucopolysaccharides which are common to both tissues. Such ideas come from several sources. First, the changes that are induced in the epithelium of the vagina in rodents under hormonal influences tend to swing the metabolism toward mucoprotein production, whereas vitamin A deficiency seems to swing it in the direction of scleroprotein production. Tissue culture experiments, though dealing with hypervitaminosis A, give some support, as do studies on the effects of hypervitaminosis A on bone formation.

The cause of hydrocephalus, which occurs in association with these other manifestations in certain animal species, is still far from settled. The thought that this might be explained on the basis of bone overgrowth and consequent pressure on the brain has been followed by evidence that in young rabbits the primary defect may be an overproduction of cerebrospinal fluid (69). It would be of interest if the choroid plexus, another epithelial structure, might be shown to manifest dysfunction or morphologic change in avitaminosis A. In this same connection, there are two clinical reports that merit consideration: one of Cornfield and Cooke (70), in which they observed hydrocephalus in a 5-month infant which had been on negligible intake of vitamin for 3 months, with rapid recovery after vitamin A therapy; and one of Bass and Caplan (77), describing development of hydrocephalus in an 8-month child while hospitalized for blurring of the cornea (with no specific mention of xerophthalmia), with recovery after 10 days of vitamin A therapy. The onset of the condition and the recovery are so rapid that one could scarcely explain them on the basis of bone changes.

A brief word about the congenital anomalies first observed by Hale (72) in pigs in 1933—which included hare-lip, cleft palate, accessory ears, subcutaneous cysts and various malformations of the eye, and called attention to the possible effect of maternal vitamin A deficiency on the developing fetus. There are the studies of Wilson and Warkany (73, 74) on fetuses of vitamin A-deficient rats, showing a large variety of defects involving various components of the eye, the heart, kidneys, and other segments of the genitourinary system. Some of these are not specific for avitaminosis A; of particular interest is the observation that during late gestation one can observe keratinizing metaplasia in certain fetal epithelia. In view

of the occurrence of congenital xerophthalmia, to which Oomen referred this morning, it would be interesting to know more about the status of epithelial tissues in late-term human fetuses under comparable situations.

By way of summary, we can state that there is a large fund of experimental information relating reasonably well to a much more limited fund of information from human pathology which specifically relates vitamin A to the normal functional and structural state of epithelial tissues of various types. Experimentally, there is a similar relationship demonstrated for the bone-forming tissues. We have as yet no real counterpart of the latter in human pathology, but it is something which certainly should be sought for where opportunity permits. We still lack a sufficient understanding of many of the experimental findings, particularly the hydrocephalic state in animals. There are only a few glimmerings as to what type of metabolic disturbance might be involved in the epithelial and bone-forming tissues. There is some hope that the application of newer technics (electron microscopy, cytochemistry and other methodology) may bring to us a more accurate picture of how vitamin A functions in cellular metabolism. Not that this will help materially in solving some of the world problems regarding vitamin A, but it will be a great source of satisfaction to those who are interested in the more basic functions of this important vitamin.

ANDRUS: I had the opportunity several years ago to examine some A-deficient tissues from cats, used in a study conducted by Dr. Stanley Gershoff of our laboratory. The chief symptoms were weight loss, weakness of the hind-quarters, and serosanguinous conjunctival exudates. We were not able to demonstrate either skeletal or nervous changes, grossly or histologically. The serosanguinous exudates correlated but poorly with the histologic eye changes, which consisted of squamous metaplasia, most commonly of the palpebral conjunctiva. The total number of vitamin A-deficient animals was 15, ranging in age from 3 to 10 months. There were three clear-cut instances of keratinizing metaplasia of the cornea, in one instance with keratomalacia and panophthalmitis. The lesions, however, that I was most interested in, with reference to what Oomen has said, were pancreatic in location, and were present in 6 out of the 15 animals. The change in the early stages seemed to consist of a loss of zymogen granules and/or a loss of cytoplasmic basophilia. In some instances the loss of basophilia with preservation of the zymogen granules made a striking picture histologically. In any case, the cell, in addition to losing these two characteristic staining features, seemed to decrease in size, and there were certain nuclear changes, such as increase in size, changes in the nucleoli and chromatin, the end result being that the cells took on a ductlike rather than acinar appearance. Inflammation was very scarce, only an occasional animal showed lymphocytic infiltration. Occasionally we did see a duct containing a few polymorphonuclear leucocytes. As to interpreting these changes, we were at quite a loss to do so. We assumed at first that these changes were probably secondary to ductal changes, i.e. obstruction due to metaplasia with ensuing atrophy. Serial sections did not reveal ductal changes sufficient to explain the acinar alterations, and we ended up by calling the latter by the rather vague, noncommital term of dysplasia. We did not think it too likely that this process might represent squamous metaplasia of the acinar epithelium. Wolbach did describe squamous metaplasia of the salivary acinar tissue, and metaplastic changes in the salivary ducts have certainly been described. There is at least one human case reported of a child with ductal changes in the pancreas but without acinar change. One other odd curiosity in these cats was squamous metaplastic cysts in the lung. These were subpleural, very superficial, and were easily spotted grossly, being a centimeter or more in size. Essentially they resembled epidermal inclusion cysts with great accumulation of keratohyaline. This change did not seem to be often complicated by infection, whereas the much more commonly seen squamous metaplasia of the trachea and bronchi led to serious complications, and several of the cats died of massive empyema or mediastinitis. I would presume from what Oomen said this morning that this change in the pancreas is quite unlike the human cases he cited from the literature.

FOLLIS: I thought it might be of interest

to present some material for which I'm indebted to Dr. Huntington Sheldon (75), who did this work while he was at the Karolinska Institute in Stockholm. This has to do with an electron microscopic study of the changes in the cornea as a result of vitamin A deficiency in mice. As one goes from the basal layer up into a mid-layer in the vitamin A-deficient animal, the normal mitochondria begin to fill up with an electron-dense material. The change coincides with the appearance of so called 'keratohyaline' in the cytoplasm, which apparently aggregates or appears within the mitochondria. Apparently, then, something has happened to the cell, and particularly to the mitochondria, so that they have begun to accumulate this material. This is a technical approach which gives us a little more of an idea on what's going on anatomically as a result of vitamin A deficiency.

HANDLER: Follis, is this behavior of corneal mitochondria shared by cells outside epithelial tissue? Do liver cells of the vitamin A-deficient animal look this way?

FOLLIS: They haven't been examined.

RAMALINGASWAMI: Mr. Chairman, I think I should be able to save some time for you because a good bit of what I wanted to say has been covered in the discussion this afternoon. I think it is perhaps appropriate to open the discussion on the structural effects of vitamin A deficiency by a reference to a statement by Wolbach which runs as follows: "The association of biochemical systems with structural details within cells is a biological goal of the greatest importance. Success with a few systems in which vitamins are operative does not seem impossible of achievement and should carry us far towards a conception of the anatomy of life."

In this presentation I would like to show you the results of some of our work which really relates to experimental studies in animals, but which was designed to understand the etiology and mechanism of production of dermal and ocular lesions which are commonly seen in clinical practice and which are attributed to vitamin A deficiency. It is unnecessary for me to go into the historical background, which has been covered adequately this afternoon, except to say that the work of Frazier and Hu (60), Lowenthal (62) and Nicholls (61), and of others that followed them, created an impression in the mid-thirties that phrynoderma or follicular hyperkeratosis is the result of vitamin A deficiency. This impression has permeated so much into public health practice that Young (76) states in 1947 as follows: "Phrynoderma of vitamin A deficiency, having become so widely advertised, everyone in examining recruits now runs his hands over the elbows and hip joints to look for the telltale nutmeg grater-like surface". There has been considerable controversy about the relationship of phrynoderma to vitamin A deficiency. In an excellent review, Stannius (77) gives no quarter to vitamin A deficiency in the etiology of phrynoderma, and goes to the extent of suggesting lack of soap as one of the etiological factors! Nutritional factors other than vitamin A deficiency obviously require consideration, and it might be just as well to remind ourselves at this stage that not all cases of follicular hyperkeratosis need be of the same etiology. The skin is known to respond in the same way to a number of widely different stimuli. The pilosebaceous follicle is apparently subject to a number of influences, nutritional and hormonal, and responds to them in similar fashion.

Patwardhan has already referred to the clinical observations and indicated the possible significance of fat deficiency in the etiology of phrynoderma. In 1949, Dr. Sinclair and I (78) began to study the structural alterations in the skin of the rat in vitamin A, essential fatty acid, and pyridofine deficiency states, and compared them with the lesions in phrynoderma. In the case of vitamin A deficiency, we used not only 21-day-old rats but also 6-week-old ones, in view of the fact that phrynoderma occurs most commonly in adolescence. We produced acute and chronic deficiency states and had calorie controls.

In none of the experiments dealing with vitamin A deficiency did we observe lesions in the skin which could be considered comparable to those seen in phrynoderma. In phrynoderma, one gets an elevated papular lesion, a plugging of the mouths of hair follicles, with dense keratotic masses in concentric layers, and/or hyperplasia of the epithelium lining the plugged follicles. The

epidermis in between the follicles also participates in such a reaction. In contrast to this, the lesion in experimental vitamin A deficiency is characterized by an atrophic change. The subcutaneous fat disappears, the sebaceous glands atrophy and disintegrate, the epidermis is atrophic and the upper third of the hair follicles is markedly dilated and contains loose keratin. The follicular lesion thus produced results in the formation of crater-like lesions rather than elevations.

While on the subject of vitamin A deficiency, I should like to comment on Oomen's observations in Indonesia on the association between vitamin A deficiency and vesical calculus. Sinclair and I observed concretions in the urinary bladder in vitamin A-deficient animals. They were seen more frequently in animals maintained on deficient diets for prolonged periods. However, I would hesitate to relate these lesions to those seen in man in calculus formation. Vesical calculus disease is a common condition affecting children in certain defined areas in India and Thailand. From our personal experiences in both these areas, it appears that this condition is not associated with clinical manifestations of vitamin A deficiency.

In fat deficiency, the induction of the syndrome and the skin lesions were in sharp contrast to those of vitamin A deficiency. The classical picture of Evans and Burr has been produced. The structural changes take weeks to appear and the animals survive for months with the skin lesions. There was a generalized acanthosis and hyperkeratosis of the epidermis, with plugging of the mouths of the hair follicles with dense concentric layers of keratin and hyperplasia of the lining epithelium of the follicles. The sebaceous glands were hypertrophic, presumably due to obstruction to outflow of sebum by the keratotic plugs in the hair follicles. Even after months of fat deficiency the subcutaneous fat was still preserved. It is clear from these illustrations that the skin changes in fat deficiency in the rat clearly resemble those seen in phrynoderma, and that they are quite different from those seen in experimental vitamin A deficiency.

PLATT: As Oomen says, I've stated my views in a chapter of a text book on tropical dermatology (24). I think we may be using different names and talking about different clinical entities. To me folliculosis is different from follicular keratosis. I'm not sure if follicular keratosis is the same as phrynoderma, which is a term I don't use. Phrynoderma probably is follicular keratosis, though the tips of the horny spines from photographs appear to be flattened. I saw several cases of follicular keratosis in China before I went to Africa. When I got to Africa, I saw many dry skins with lesions of the follicles; but it was so unlike what I was accustomed to see in China that I invented a simple name for it. I called it permanent goose flesh, P.G.F., for short. I still don't know what it is, and nobody has bothered to investigate it. It may be the same thing as follicular keratosis but until we find out what it is, I shall assume that it's different. What I mean by follicular keratosis is well shown in illustrations in my chapter on the skin in malnutrition in Simon's book (24). In a biopsy specimen after treatment throughout the epidermis there are mitotic figures. Mitosis is usually confined to the stratum germinativum. Mitosis distributed throughout the epidermis has also been observed by Wolbach in animal skins in the course of deprivation of vitamin A. This seems then to occur during repletion of the animals with vitamin A. This particular patient, a youth of 17 years of age, was one of a group with similar lesions; they were all Northerners from Peking, the same place as Frazier and Hu (60) saw follicular keratosis. They continued to eat the foods they had in the North, i.e., a wheat flour preparation, a sort of steamed pudding. Now I think that may be important because, as I understand Hugh Sinclair's views on essential fatty acids (EFA), he would maintain that a diet with wheat flour as a staple would furnish all the EFA needed in the diet if the EFA's had not been destroyed by so-called 'improvers'; and I doubt very much if 20-odd years ago the Chinese were using "improvers'. So that according to Sinclair's notion on this subject, they should have had enough EFA in their diets. They were getting plenty of vegetables. The hospital diet was a generally poor one and was unlikely to account for the recovery of this patient. I gave 100,000 IU of vitamin A daily

for 2 weeks, and then reduced the dose to 18,000 daily. After 10 days many of the spinules had fallen out; the skin became moist and greasy; the hair improved in appearance and acquired a definite luster. The improvement was maintained, until at the end of about 6 weeks the roughness of the skin and the spinules almost completely cleared. I don't think this evidence is necessarily opposed to the findings of Patwardhan; it may well be that we are dealing with similar end results from different disorders due to insufficiency of different food factors.

PATWARDHAN: You will see from the picture from my monograph (55) which I shall pass round that the condition which I was describing and one which Platt described are essentially the same. I know that Platt and I have been holding different opinions on the etiology of phrynoderma for the last 7 years. Platt's experience in the treatment of phrynoderma in 1936–37 refers to an era when vitamin A was not known. It was usual to administer vitamin A in oil solutions, so that one did not know whether vitamin A or the oil which contained essential fatty acids was responsible for the cure. Now that synthetic vitamin A has become available, one can administer it as a water-miscible preparation, as we and Scrimshaw have done, to assess the effect of vitamin A on phrynoderma. No amount of argument at this table is going to settle this issue. Those who question the role of essential fatty acids in the etiology of phrynoderma should at least make an attempt to settle this question by conducting comparative experiments with oils rich in essential fatty acids and with vitamin A. The problem is not of great practical importance, but it is of fundamental importance, because we seem to have gotten stuck at one particular phase of our understanding of the manifestations of vitamin A deficiency. I feel that scientists like Platt, Darby, and others, who have a chance to work in the tropics where they must encounter phrynoderma, should convince themselves concerning its etiology. Pending that, we need not take extreme positions on this question. I am prepared to concede, as Ramalingaswami said, that we may be dealing with a condition which is not fully understood. It may result from a deficiency of essential fatty acids, a disturbance in fatty acid metabolism, or a combination of a deficiency of vitamin A and essential fatty acids. The relationship between fat metabolism and vitamin A pointed out by Ramalingaswami earlier in his presentation must be kept in mind. I suggest that cases of xerophthalmia and phrynoderma may have different etiologies. I submit that this problem should be included in our recommendations as one of those requiring further investigation.

SCRIMSHAW: Based on the negative results of giving vitamin A and on the knowledge of the success in India in treating this condition with fat, we devised the experiments to which I referred yesterday in connection with goiter to determine specifically the effect of fat on follicular hyperkeratosis. Among school children, where the frequency ran between 45 and 55%, the administration of 48 gm of lard daily, 5 days a week for 56 weeks, of hydrogenated cotton-seed oil, 48 gm daily, 5 days a week for 56 weeks, and of the same amount of cotton-seed oil for 34 weeks, all failed to have any detectable effect on the follicular hyperkeratosis.

HIGGINSON: A lesion which has been described in the Bantu by our clinical colleagues has sometimes been referred to as follicular keratosis but which on careful clinical examination shows peri-follicular hemorrhages. These may be difficult to note on a pigmented skin and may only be observed after histological section. Now this condition appears very similar to what Platt calls 'permanent goose flesh'. It responds to ascorbic acid administration and it should be considered in the differential diagnosis of that skin condition.

PLATT: I would like to ask Ramalingaswami what skin changes were found in his A-deficient monkeys.

RAMALINGASWAMI: The changes in the monkeys were similar to those in A-deficient rats. Regarding Higginson's point, the occurrence of follicular hyperkeratosis in scurvy is recognized. However, follicular hemorrhages are not observed in cases of phrynoderma.

MCLAREN: I'd like to get away from the skin. If hydrocephalus in rabbits deficient in vitamin A is due to over-secretion of

cerebrospinal fluid by the choroid plexus, one would expect to find evidence of raised intro-ocular pressure, glaucoma, in these rabbits in view of the close similarity of cerebrospinal fluid and aqueous humour as to mode of production and composition.

V. Biochemical Aspects of Vitamin A

Handler: Clearly, this group would like an explanation in molecular terms of the manifestations of vitamin A deficiency. Unhappily, I am not aware of any vitamin deficiency state which can presently be explained in these terms, except for unique or specific aspects. In this case, the magnificent work of Kuhne, Selig Hecht, and Wald, and many of the others who have contributed to elucidation of the mechanism of rod vision requires no apology. Nor, does it require any exposition at this meeting. It has been described well in many textbooks and in recent reviews but has one feature to which I shall return. Thus, whereas we do not have explanations for all the other lesions which have clamored for your attention today, for at least one of them, night blindness, we have a wonderfully complete explanation.

The literature on vitamin A is huge and any attempt to review it would be a waste at this time. There are only a few questions to which I think we might address ourselves. The first of these relates to the absorption of the carotenes and vitamin A precursors in the intestine. Strangely enough, at this time, we are still not quite certain where it is that carotenes are converted into vitamin A. There is ample evidence that the intestinal mucosa conducts this transformation but there is other evidence that suggests that the liver is also capable of such transformation. In this regard there is the repeated observation that in severe liver damage in man, damage unrelated to nutritional deficiency, the ability to perform the beta-carotene to vitamin A conversion is seriously impaired. Indeed, this has been used as the basis of a reasonably satisfactory liver function test in alcoholic cirrhosis, in infectious hepatitis and biliary disease. To this observation I would like to call the attention of those of you who have been concerned with the absorption or utility of carotene in malnourished individuals, particularly infants with kwashiorkor. If their livers are functionally damaged, they would be unable adequately to perform this particular conversion, and carotene would not be as useful to them as would vitamin A. However, the mechanism of this conversion of carotene to vitamin A has been entirely elusive. There is no known enzyme capable of performing this scission of the molecule to its two halves. There is another mystery in this connection. Beta-carotene is twice as useful as alpha-carotene in rat nutrition, suggesting that both halves of the molecule are precursors of vitamin A. Yet in rat nutrition beta-carotene is never as useful as an equal weight of vitamin A. This suggests that the absorption of carotene, at best, is never as good as that of absorption of vitamin A itself, regardless of the conditions of administration. The reasons for this are not apparent.

There is much literature which relates to the necessity for adequate fat absorption while absorbing carotene or vitamin A. All circumstances which lead to steatorrhea also impair vitamin A and carotene absorption. There is also the large body of literature which indicates that the stability of vitamin A and beta-carotene are, in part, dependent on the concomitant presence of anti-oxidants, usually alpha-tocopherol, which protect carotenoids against autoxidation, particularly in the presence of lipoxidase from plant sources. Relatively little attention seems to have been given to this problem in human nutrition, although rat nutritionists who have sought to quantitate the biological response to carotene or vitamin A have been acutely aware of this problem.

Until just a short time ago, biochemists were beginning to be concerned that the physiological role of none of the fat-soluble vitamins could be explained in terms of specific reactions in which they participate, and that, like hormones, we would have to understand the role of fat-soluble vita-

mins in terms of the behavior of intact cells. In the last year or two it has become apparent that vitamins E and K probably participate in oxidative phosphorylation in mitochondria and, if this be true, there arises the hope that vitamins A and D might also participate in reaction systems which a biochemist might hope to understand. But we must remain cognizant that this may not be the case. We must not forget that vitamin A is present only in animal material. This bodes ill for the prospect of our understanding its role. Our successes in establishing the functions of water-soluble vitamins relate to the fact that all of these participate in reactions of such basic significance that they occur in all living forms. To my knowledge, no bacteria contain vitamin A and few of them contain carotenoids of any sort. The carotenoids distributed in the plant kingdom are usually restricted to the green portion of the plant and there have been many speculations as to their role in the initial photochemical processes of photosynthesis. It is rather interesting to note that the only thing we do know about vitamin A in the physiology of animals also relates to a photochemical process. Animals have adapted for vision a molecule which may serve a similar photorecepter role in plants. But if vitamin A is uniquely an animal material, it probably participates in a process which is unique to animals. This is simply a teleological guess. But in that case, our problems in learning its role in cells other than those of the retina are likely to be rather difficult. Thus, it may be concerned with mucopolysaccharide or mucoprotein formation. This suggestion arises from consideration of the lesions which are found in epithelial tissue and in bone of deficient animals. Bone is seriously dependent upon the formation of mucoprotein for continued growth. The fact that bone growth ceases before the growth of many of the soft tissues of the animal may indicate that this is the kind of lesion for which we should seek. And here we can only recognize that one wastes much time and effort unless the right experiment is done at the right time. If you attempt it too soon you'll probably learn nothing. In this case, I suspect that we don't really know enough to tackle the problem of the mechanism in which vitamin A participates. If, for example, it does really relate to mucoprotein synthesis, we know almost nothing about the chemical mechanisms involved in mucoprotein synthesis. We know almost nothing about synthesis of proteins in general and, for mucopolysaccharides, we have just begun to develop the methods necessary to tackle the problem. When these methods are available and the information is available, then will be the time to see whether vitamin A can affect them. This is always the case. I wonder how much effort has been wasted over the years by investigators seeking explanations with the information at hand, in the hope that sufficient background information was already at hand. A case in point is the recent observation that the vitamin A deficiency animal seems to have an impaired ability to convert pyruvate to glycogen in the liver. I'm sure that the data are correct, but I would also be almost certain that this phenomenon is unrelated to the usual signs of vitamin A deficiency. This is a purely personal reaction and needn't be taken too seriously.

Follis showed us mitochondria from a deficient cornea which are filled with a material which pathologists label with a term which a biochemist won't even speak. Whereas biochemists know something about liver mitochondria and about kidney mitochondria, they know nothing about corneal mitochondria. They can only assume that mitochondria are the powerhouses of the cell wherever you find them, the place in which the citric acid cycle and its attendant oxidative phosphorylation is conducted. One cannot help but wonder whether or not the process described by Follis is uniquely related to mitochondria in the tissues which are sensitive to vitamin A deficiency or whether this is a general phenomenon. Here we might point out that there is some information concerning the distribution of vitamin A in liver. Liver homogenates separated by differential centrifugation into the clear supernatant, microsomal, mitochondria and nuclei fractions have been analyzed for the distribution of the vitamin A. The bulk of the vitamin A, in stock-fed rats, was located

in the 'supernatant' fraction. Relatively little was in the mitochondria, or other particulate fractions. This is somewhat surprising, since the mitochondria and microsomes between them contain almost all the lipid of the liver cell and are where I would have expected the vitamin A to be in simple solution, if nothing else. Accordingly, the observation which Follis mentioned came as something of a surprise to me, in that if vitamin A plays any role in the metabolism of the liver, according to its distribution, it shouldn't be in the mitochondria. The fact that most of this vitamin A is in 'storage' may obscure the role of the 'functional' material.

Finally, this brings us to the role of vitamin A in the eye. It seems pointless to spend much time on this phenomenon, which has been so beautifully described by Wald, who has contributed so much of the information. The point to which I would like to call your attention is the fact that in this photochemical process there is a profound alteration in the structure of the side chain of the vitamin A molecule. The material in rhodopsin itself has the side chain which includes two *cis* double bonds. The material which comes away from the protein, after it has accepted photons, has an all-*trans* configuration. This all-*trans* compound cannot get back onto the protein. This requires the activity of an enzyme which Wald has called an isomerase. This isomerizes the double bonds of the sidechain. The enzyme is located in the retina, and there may also be such activity in liver. During this time, the period in the visual cycle in which the vitamin A has come away from the protein as an aldehyde, it is reduced to vitamin A alcohol in the *trans*-configuration. It is only during this period that vitamin A can escape from the retina. It is the fact that it can escape while it is unbound and must be isomerized and reoxidized before it can be rejoined to the protein, which presumably makes for the fact that there is a demand for vitamin A in the retina to compensate for a loss. This is presumably reflected in the tale we heard today of what happens to sailors who have been on vitamin A-low diets. After a day at sea in which the total number of photons absorbed in their retinae has been at a maximum, the vitamin A supply of the retina has been reduced sufficiently as to impair vision. In the cones there is another opsin, a protein which also binds retinine. The absorption spectrum of this material differs slightly from that of rod rhodopsin. This has afforded no satisfaction to those of us who want to understand color vision.

The fact that animals continue to require vitamin A to some extent even in adult life implies that, somehow, we dispose of vitamin A. Clearly we do not do so rapidly. If there were an active disposal system which would oxidize vitamin A rapidly, it would be impossible to give substantial doses which can be stored in the liver. No enzyme systems are known which destroy vitamin A or in any way impair its usefulness. However, labeled material given to animals has resulted in the production of $C^{14}O_2$ in the exhaled air, so that slowly but surely vitamin A can be destroyed, ultimately leading to carbon dioxide. It is this fact which makes it an indispensable nutrient—something which we continue to require.

VI. The Pathogenesis of Hypovitaminosis A

McLaren: Pathogenesis deals with the methods by which lesions are produced by etiological factors. The lesions, under pathology, and the etiological factors, in the sections of clinical aspects and epidemiology, have been adequately covered already and our knowledge is quite extensive on these facets of hypovitaminosis A. Pathogenesis, whereby the two are linked, on the other hand is almost virgin soil. Indeed it would not be difficult to defend the view that the mode of production of the lesions resulting from a deficiency of vitamin A forms the greatest gap in our knowledge in vitaminology at the present time. It is my difficult task to bring together the causal factors on the one hand and the tissue changes on the other, and to attempt to suggest, I can do no more, how they may be related.

I have divided my subject matter into two parts, leaving the consideration of certain important unsolved problems in vitamin A metabolism until later and dealing firstly with: Ways in which a deficiency of vitamin A may arise in the body.

Lowered dietary intake is, of course, the obvious and most commonly operating factor. Hypovitaminosis A can, however, arise in the absence of a dietary defect, when other explanations have to be sought. There is need for work to be done on the daily supply of vitamin A, amongst other constituents, in the breast milk of malnourished mothers. While Moulemans and de Haas (79), for instance, showed that the concentration of vitamin A was very low in the breast milk of Indonesian women, Darby and myself, when in Java last year, considered that equally important may be a low volume of milk. This last factor probably varies considerably in different races.

A less obvious source of intake of the vitamin is the transplacental transfer to the fetus, where a comparison of early and more recent studies of neonatal liver reserves suggests that the level of maternal stores may considerably affect those laid down by the fetus (78, 79), although this does not seem to be true for the rat (80).

Impaired intestinal absorption may result in a secondary hypovitaminosis A in such disorders as the steatorrheas, fibrocystic disease of the pancreas and other affections of that organ, biliary insufficiency and small gut insufficiency following operation, etc. Whipple's disease or intestinal lipodystrophy is a disorder of the intestinal lymphatics and in view of the evidence (81–83) that the lymphatics are of more importance than the portal vein for the absorption of the vitamin, hypovitaminosis A might be expected to be a prominent feature of the syndrome and this is supported by the low plasma levels of vitamin A and carotenoids and very poor absorption in a case described by Story and Sagild (86).

In protein malnutrition the pancreatic fibrosis and intestinal lesions commonly found and the associated reduction of pancreatic and intestinal enzymes may also adversely affect absorption of vitamin A as is suggested by work from Central America (87). Furthermore, deficiency of vitamin A may itself further impair absorption, as intestinal lesions are common in a variety of deficient animals. Infestation with intestinal parasites may also interfere with absorption of vitamin A. *Ascaris lumbricoides* is known to impair protein absorption (88), and its frequent association with hypovitaminosis A as well as kwashiorkor may be more than coincidental.

Other factors which are known to influence absorption of both preformed vitamin and provitamin are level and type of protein in the diet (89), fat in the diet (90), protective action of tocopherols and other anti-oxidants, bile secretion (91–92), and immaturity (93).

The small intestine is now known to be the main site of conversion of the provitamin into vitamin A, although in those animals, including man, which have circulating carotenoids it is quite possible that further conversion may take place in the liver. In view of the fact that the chief dietary source of the vitamin in large parts of the world is vegetable, inefficient carotene conversion may be one of the most important of all factors but evidence is needed to show that this is so. Some experience of Jonxis (94) during and after the Second World War in Holland suggests that carotene utilization may be especially poor in young children.

It is almost certain that both vitamin A alcohol and carotene are carried in the blood loosely bound to protein, probably albumin. It is therefore possible that in any of the many conditions in which plasma albumin is lowered this process might be interfered with. Even so, liver storage may not suffer, as it has been mentioned already that the lymphatic route from intestine to liver is probably more important than the portal vein. It might, however, affect distribution to the rest of the body, and this could account for certain anomalous personal results with rats deficient in both protein and vitamin A, and of Guilbert and his colleagues (95, 96) when prolonged deficiency in cattle and poultry resulted in clinical and pathological signs of hypovitaminosis A while the liver still contained traces of the vitamin.

Storage takes place mainly in the liver, but when stores are low more may be present in the kidneys. The vitamin is normally stored in the reticuloendothelial cells of the liver and in those conditions which damage these cells storage might be expected to be affected. It is possible that some of the features of the group of diseases known as the lipoidoses are really manifestations of such a secondary hypovitaminosis A.

Diseases affecting the liver, other than those specifically damaging the reticuloendothelial cells, may or may not affect the storage of vitamin A. For instance, the evidence from experimental work is that fatty infiltration of the liver does not have an adverse effect on the storage of vitamin A (97), but in human cirrhosis there may be almost no vitamin in the liver, and Haig and Patek (98) found the blood plasma vitamin A level a useful index of the stage of this condition.

Other factors of importance in liver storage are the daily dosage, for animal work has shown that the efficiency of liver storage is proportional to this. Moreover, vitamin E and other antioxidants improve storage. Finally, stores are greater in the female than in the male.

Strangely enough, kidney stores are greater in the male than the female (99). Histological differences in the liver and kidneys and in the two sexes may account for these findings in view of some work on mice (100).

This leads me to the second part of my subject: Some unsolved problems of the intracellular role of vitamin A.

Outside of the retina, where the participation of vitamin A in vision is well known, we have almost no knowledge of its physiological role in other tissues, although it soon becomes conspicuous by its absence. However, as Wald and Brown (101) have suggested, the major role of sulphydryl (—SH) in the synthesis of rhodopsin in the retina, does suggest that this most reactive group in the whole of biochemistry may be found in other tissues in combination with vitamin A or a derivative of it, just as in the retina the sulphydryl groups of the protein opsin react with retinene, which is vitamin A aldehyde. Furthermore, retinene is converted into vitamin A by the sulphydryl enzyme alcohol dehydrogenase.

Recently, studies have been made of sulphydryl groups in the normal skin, after local application of the vitamin, and also in hypovitaminosis A (102–104). However, no new light on the problem of the relationship of vitamin A and —SH groups has come from this work and at present all we can do is surmise that, as we know is the case in the retina, so also in the epithelia where hypovitaminosis A manifests itself, —SH groups and vitamin A are vitally concerned together.

Popper (105) extensively explored the distribution of vitamin A in the tissues by this method. Even in far-advanced hypovitaminosis A, long after other organs had lost their vitamin A fluorescence, it is still observed in the retina. However, in both humans and rats, there was complete absence of fluorescence from the normal cornea, and this was true even in hypervitaminotic rats. It has, of course, been suggested that the state of combination of the vitamin in the tissues determines the property of fluorescence, but it is just here that one of the main hiatuses in our knowledge of the vitamin lies. A reinvestigation with more recent spectrophotometric methods or autoradiography should clarify this point.

Morton and his colleagues (106–108) discovered two substances, which they called 'A' and 'C'. These substances have characteristic absorption spectra, and their presence and concentration in the liver and certain other organs is dependent upon the vitamin A status. These workers have also found several other unknown substances by spectroscopic examination in hypovitaminosis A.

Most of this work was done on the rat, and very recently it has been shown (108) that no rise occurs in the concentrations of SA and AC in the domestic fowl suffering from hypovitaminosis A. It is not yet known whether these substances accumulate in human hypovitaminosis A, but suggestive that they may do so is the similarity of the symptoms in man and the rat, both readily developing xerophthalmia,

while manifestations are different in the fowl and eye lesions do not occur.

One of the discoveries of Fell and Mellanby (109), using tissue culture methods, was that vitamin A has a direct effect in causing epithelial cells to change from squamous to mucus-secreting type. It has yet to be shown that the reverse process is possible, i.e. that by using a medium containing plasma from vitamin A-deficient animals mucus membranes may be changed to squamous epithelia.

Mason and Ellison (110) seem to have been the first to suggest that in hypovitaminosis A there is a defective formation of mucin, made up of glyco-proteins, in epithelial cells, with a corresponding increase in keratin, which is composed of albuminoids. Here, once again, sulphydryl groups appear to come into the picture, for the oxidation of —SH to disulphide (—SS) is part of the keratinization process.

In the deepest layer of cells of the stratum granulosum of the skin the cytoplasm contains granules which stain deeply with hematoxylin and are known as keratohyaline granules. The cells of the epidermis begin to die in the stratum granulosum and the presence of these granules seems to indicate the first stage in the transformation of living cells into keratin. Leber (16) is given credit by Mori (11) for being the first to describe these granules in the intermediate layers of the cornea undergoing the keratinizing metaplasia characteristic of hypovitaminosis A.

As Follis mentioned, recently Sheldon and Zetterqvist (75) have shown by the use of the electron microscope that in the cornea of the vitamin A-deficient mouse the keratohyalia granules are derived from degenerating mitochondria. This calls to mind the opinion expressed more than 20 years ago by that very great worker in this field, Wolbach (111), with which I may fittingly end, "Vitamin A may be solely concerned in maintaining an apparatus within cells and not in the chemical processes for which the apparatus is necessary."

Olson: I should like to address myself to the concluding statement of McLaren's paper. It is the citation of Dr. Wolbach's belief that the fat-soluble vitamins play some structural role in the cell. Some now working in this field are convinced that fat-soluble vitamins function as part of the intracellular lipoproteins. This is certainly true of vitamin E, which is found in the mitochondria and which, in the form of its quinone, may participate in electron transport, as Handler mentioned. In addition to a possible role in oxidation phosphorylation, it appears to me that it may have a further role when stabilizing the lipoprotein matrix of the mitrocondria. In regard to the comment made about the experiments from Duell's laboratory on studies on the distribution of vitamin A in subcellular fractions from liver, I should like to point out that sucrose fractionation is rather rugged treatment for the lipoproteins of the particulates, and some of the free fat which was measured in those experiments might be artifactual. We find very little free fat in the cytoplasm of liver if suitable methods are used. The question about the absence of vitamin A in microsomes and mitochondria is a moot point; further, even under Duell's conditions he found some vitamin A in the nuclei. And it is known that the nuclei as well as the other particulates do contain appreciable amounts of lipid.

The second point I should like to make relates to the problem of conditioned vitamin A deficiency, which was touched on by McLaren. It is of course, well known that conditions that interfere with fat absorption tend to condition vitamin A deficiency. I would like to cite another kind of conditioned vitamin A deficiency which we have seen in our nutrition clinic at the University of Pittsburgh.

A young woman of 28 years came to us with the chief complaint of difficulty in night vision, and a skin lesion which was typical follicular hyperkeratosis. This woman was well nourished, had no steatorrhea, no obvious gastrointestinal disease and was otherwise in good health. She had been studied by Hecht in New York at the age of 15 when these symptoms first presented and was found to have a defect in dark adaptation. She was given intramuscular vitamin A at that time with remission of the defect in dark adaptation and some remission of the follicular hyperkeratosis. When we saw her she had not

been taking a vitamin A supplement and had a recurrence of symptoms. Her serum vitamin A was about 20 μg%, which is low in our clinic, despite the fact that her diet contained over 5000 IU of vitamin A. On oral therapy of 100,000 IU per diem her serum A came back to about 55 μg, accompanied by symptomatic improvement in night vision and some reduction of her hyperkeratosis.

What I am suggesting is that there may be some rare, possibly genetically determined, abnormalities in the absorption of vitamin A which may condition mild forms of this deficiency disease.

SCRIMSHAW: It should be noted that despite the tremendous increase in fat in the liver of the child with kwashiorkor, the usual liver function tests are not consistently altered. Furthermore, between Waterlow and Patrick (114) and work of Burch with the INCAP group (115), some 12 basic enzymes have been examined in the liver of children with kwashiorkor; of these only nonspecific cholinesterase, xanthinoxidase and amino acid oxidase have been found to be altered. There is a practical point in evaluating vitamin A activity of diets, which no one seems to have mentioned. Vitamin A activity can't be measured by total carotene, because of the many inactive carotenoids in foods, but they can't be measured by beta-carotene either, because there are a number of carotenoids in food stuffs also which contribute to the vitamin A activity of the diets such as those in rural Guatemala, which may contain as much as 80% of yellow corn, nearly half of the vitamin A activity could be missed if beta-carotene alone were determined. This is due to the fact that some corns contain more cryptoxeanthin than beta-carotene.

MACKENZIE: I would just like to make a brief comment about the distribution of fat-soluble vitamin in the organelles of cells. Up until a few years ago it was categorically asserted that vitamin K was not in the mitochondria of cells and yet now, as Handler pointed out, vitamin K is not only known to be there but there is much speculation concerning its role there. With respect to vitamin A, if I understand Handler's reference to the literature correctly, the studies were done on liver, and that if one wishes to know the role of a vitamin and its distribution in organelles that is the tissue not to use, for it is the one that stores it. In the case of vitamin A, the large storage in the liver may completely obscure the part of the vitamin A that's playing some essential function. So I think that the question of where vitamin A is inside cells when it's performing its vital role should be left open.

PLATT: Just a simple comment about systemic infections. I would assert the most likely thing that happens is that the child reduces its appetite.

SCRIMSHAW: During some recent metabolic studies in two children receiving a diet supplying 3 grams of protein per kilogram of body weight, we were astonished to find that the prodromal phase of chickenpox was sufficient in both children markedly to reduce nitrogen retention. This suggests that even mild systemic infections can have marked metabolic effects.

AYKROYD: We started with a discussion this morning of epidemiology. Out of this emerged the fact that vitamin A deficiency is an important problem in different parts of the world, particularly in Asia and Africa. We were told that within certain countries of Asia, in India and China for example, there was a difference in incidence between south and north. Differences in Africa, related to the use of red palm oil, were also mentioned. It was pointed out that vitamin A deficiency is largely a common occurrence among rice-eaters, and certain suggestions were made to account for this. It was stated that the disease is at present uncommon or unknown in the U.S.A., which would also apply to northern Europe generally. An interesting point was brought forward by one speaker, who indicated that the incidence of vitamin A deficiency might not necessarily be related to the vitamin A content of the local diet, but rather to certain social habits which might influence the feeding of young children, such as the time of weaning and the spacing of children in the family. Oomen and others noticed that the severe vitamin A deficiency states largely occur in young children, common to the age group that suffers from protein malnutrition, and that the condition in older children is in general much less serious. A reference to dark adaptation

tests may also be mentioned. These tests have proved of little value in well-fed countries such as the U.S.A. and countries in northern Europe and because everybody was in a pretty good state of vitamin A nutrition. Such tests might be more useful applied in countries in which there is more vitamin A deficiency.

With respect to the clinical aspects, Oomen gave us an extremely vivid and striking picture, and an important question which emerged from his exposé was the possible distinction, with respect to etiology, between keratomalacia and xerophthalmia. There was not, however, general agreement on that point and subsequently Oomen admitted that there is a very close association between keratomalacia and vitamin A deficiency. He and other speakers brought the association out between protein malnutrition and vitamin A deficiency. Throughout our discussions no obvious clinical association has been brought out between eye lesions due to vitamin A deficiency and lesions of other systems, such as the nervous or genitourinary systems.

From our discussions on pathology, one gained the impression that studies have so far thrown very little light on this subject beyond demonstrating some similarity in eye and skin conditions produced in experimental animals by vitamin A deficiency and changes occurring in human cases. This may perhaps be due to variations in the physiological reactions of different species to vitamin A deficiency. With respect to biochemistry, it was useful for us to have had the exposé of Handler, who stated that, except in one area of the subject, namely, the relation between vitamin A and retinal function, present knowledge of the biochemistry of vitamin A deficiency is extremely limited. He went on to tell us that the whole subject was one which is difficult to investigate from the biochemical standpoint.

Finally, McLaren gave us a catalogue of problems in this field, indicating many points at which our knowledge is defective and further research is needed.

REFERENCES

1. Aykroyd, W. R. *Current Science* 13: 149, 1944.
2. Wille, W. A. *Trans. 4th Congr. Far Eastern Assn. of Trop. Med. Batavia* 1: 245, 1921.
3. Kreiker, A. *Graefe's Arch. Ophth.* 124: 191, 1930.
4. Pillat, A. *Arch. Ophth.* 2: 256 and 399, 1929.
5. Pillat, A. *China M. J.* 43: 907, 1929.
6. Pillat, A. *Nat. M. J. China* 15: 614, 1929.
7. Yap-Kie-Tiong, *Brit. J. Ophth.* 40: 502, 1956.
8. Yap-Kie-Tiong. *Brit. J. Nutr.* 11: 158, 1950.
9. Mori, M. *Jahrb. Kinderheilk.* 59: 175, 1904.
10. Sie-Boen Lian. *Geneesk. Tydschr. Ned. Indie* 69: 1097, 1929.
11. Mori, S. *Bull. Johns Hopkins Hosp.* 33: 357, 1922.
12. Sweet, C. K. and H. J. K'ang. *Am. J. Dis Child.* 50: 699, 1935.
13. Thompson, F. A. *Trans. Roy. Soc. Trop. Med. Hyg.* 47: 159, 1953.
14. Pillat, A. *Graefe's Arch. Ophth.* 128: 361, 1932.
15. de Gouvea, H. *Graefe's Arch. Ophth.* 29: 1, 167, 1883.
16. Leber, T. *Graefe's Arch. Ophth.* 29: 3, 225, 1883.
17. Bloch, C. E. *Jahrb. Kinderheilk.* 89: 405, 1919.
18. Blegvad, O. *Am. J. Ophth.* 7: 89, 1924.
19. van Stockum, M. *Geneesk. Tydschr. Ned. Indie* 78: 856, 1938.
20. de Haas, J. H., J. H. Posthuma and O. Meulemans. *Geneesk. Tydschr. Ned. Indie* 80: 928, 1940.
21. Oomen, H. A. P. C. *Docum. de med. geogr. trop.* 5: 195, 1953.
22. Oomen, H. A. P. C. *Brit. J. Nutr.* 8: 307, 1954.
23. Nicol, B. M. *Brit. J. Nutrition* 3: 25, 1949.
24. Platt, B. S. In: Simons, R. D. G. Ph., *Handbook of Tropical Dermatology II*, Ch. 94: Clinical and biochemical features of the skin in malnutrition. Amsterdam: Elsevier, 1953.
25. de Langen, C. D. *Geneesk. Tydschr. Ned. Indie* 69: 806, 1929.
26. Senduk, R. C. E. Urinestenen te Batavia (Urinary calculus at Batavia). Thesis, Batavia, 1949.
27. de Haas, J. H. *Meded. Dienst Volksgezondheid in Ned. Indie* 1: 1, 1931.
28. Moore, T. *Vitamin A*. Amsterdam; Elsevier, 1957.
29. Zuidema, P. J. *Docum. de med. geogr. trop.* 7: 229, 1955.
30. Blackfan, K. D. and S. B. Wolbach. *J. Pediat.* 3: 679, 1933.
31. Frank, M. *Med. Klin.*, No. 15, 1935.
32. Uotila, U. and P. E. Simola. *Virchow's Arch.* 301: 523, 1938.
33. de Haas, J. H. and O. Meulemans. *Lancet* 1: 1110, 1938.
34. Pillat, A. and A. Fuchs. *Klin. Monbl. Augheilk.* 68: 646, 1922.
35. Oomen, H. A. P. C., Rochilin Prawirowinoto and L. E. Latuasan, Berita Kementerian Kesehatan Republik Indonesia 5: okt. Makanan, Kesehatan dan Keadaan Gizi Anak Ketjil (Food and health in average Djakarta toddlers), 1954.
36. Sie-Boen Lian. *Geneesk Tydschr. Ned. Indie* 77: 3283, 1937.

37. Mulock Houwer, A. W. *Handl. 7e Ned. Ind. Nat. Wetensch.*, 1935.
38. Tijssen, J. *Ned. Tydschr. v. Geneesk.* 78: 5452, 1934.
39. Gomperts, C. E. *Geneesk. Tydschr. Ned. Indie* 80: 1192, 1940.
40. Tijssen, J. *Voeding* 2: 45, 1940.
41. Oomen, H. A. P. C. *Docum. de med. geogr. trop.* 9: 357, 1957.
42. van Veen, A. G. and J. C. Lanzing. *Geneesk. Tydschr. Ned. Indie* 80: 514, 1940.
43. Metivier, V. M. *Am. J. Ophth.* 24: 1029, 1941.
44. Klerks, J. V. The assessment of the nutritional status of Indonesian school children, 1956.
45. Courbieres, M. Vitamin and avitaminosis A. Thesis, Lyons, 1935.
46. Hoogenkamp, P. A. Development, diet and health in children of South Borneo. Thesis, Amsterdam, 1956.
47. de Haas, J. H. and O. Meulemans. *Geneesk. Tydschr. Ned. Indie* 78: 847, 1938.
48. Oomen, H. A. P. C. and S. H. Malcolm. Nutrition and the Papua child. A study in human welfare. Technical Paper, South Pacific Commission, Noumea, 1958.
49. Aykroyd, W. R. and B. G. Krishnan. *Indian J. Med. Res.* 23: 741, 1935.
50. McLaren, D. S., Jr. *Trop. Ped.* 2: 135, 1956.
51. Roels, O. A., *et al. Doc. de med. geogr. trop.* 10: 77, 1958.
52. Lambert, R. A. and A. M. Yudkin. *J. Exper. Med.* 38: 25, 1923.
53. Gopalan, C. *Indian Med. Gaz.* 82: 16, 1947.
54. Menon, P. S., P. G. Tulpula and V. N. Patwardhan. *Ind. J. Med. Res.* 38: 173, 1950.
55. Patwardhan, V. N. *Nutrition in India*, Bombay: 1952.
56. Goldschmidt, M. *Arch. Ophth.* 90: 345, 1915.
57. Wolbach, S. B. and P. R. Howe. *J. Exper. Med.* 42: 753, 1925.
58. Wolbach, S. B. and P. R. Howe. *Arch. Path. Lab. Med.* 5: 239, 1928.
59. Wolbach, S. B. and P. R. Howe. *J. Exper. Med.* 57: 511, 1933.
60. Frazier, C. N. and C. K. Hu. *Arch. Int. Med.* 48: 507, 1931.
61. Nicholls, L. *Indian M. J.* 68: 681, 1933.
62. Loweenthal, L. J. A. *Arch. Dermatol. Syphilol.* 28: 700, 1933; *East African M. J.* 10: 58, 1933.
63. Hetler, R. A. *J. Nutrition* 8: 75, 1934.
64. Mellanby, E. *J. Path. Bact.* 38: 391, 1934.
65. Moore, L. A. *J. Nutrition* 17: 443, 1939.
66. Moore, L. A. *J. Dairy Sci.* 24: 893, 1941.
67. Mellanby, E. *J. Physiol.* 101: 408, 1943. 105: 382, 1947.
68. Wolbach, S. B. and O. A. Bessey. *Arch. Path.* 32: 689, 1941.
69. Miller, J. W. and D. H. M. Woollam. *J. Neurol. Neurosurg. Psychiat.* 19: 17, 1956.
70. Cornfield, D. and R. E. Cooke. *Pediatrics* 10: 33, 1952.
71. Bass, M. H. and J. Caplan. *J. Pediat.* 47: 690, 1955.
72. Hale, F. *Heredity* 24: 105–106, 1933.
73. Wilson, J. G. and J. Warkany. *Am. J. Anat.* 83: 357, 1948.
74. Wilson, J. G. and J. Warkany. *Am. J. Anat.* 85: 113, 1949.
75. Sheldon, H. and H. Zetterqvist. *Bull. Johns Hopkins Hosp.* 98: 372, 1956.
76. Young, W. A., A. G. Farr and A. McKendrick. *E. African M. J.* 24: 127, 1947.
77. Stannus, H. S. *Proc. Roy. Soc. Med.* 38: 337, 1945.
78. Ramalingaswami, V. and H. M. Sinclair. *Brit. J. Dermatol.* 65: 1, 1953.
79. Meulemans, O. and J. H. de Haas. *Geneesk. Tijdschr. Ned.-Indie* 25: 1938.
80. Wolff, L. K. *Lancet* 223: 617, 1932.
81. Lewis, J. M., O. Bodansky and L. M. Shapiro. *Am. J. Dis. Child.* 56: 503, 1943.
82. Dann, W. J. *Biochem. J.* 26: 1072, 1932; 28: 634, 1934.
83. Drummond, J. C., M. E. Bell and E. T. Palmer. *Brit. M. J.* 1: 1209, 1935.
84. Eden, E. and K. C. Sellers. *Biochem. J.* 44: 264, 1949.
85. Popper, M. and B. W. Volk. *Arch. Path.* 38: 71, 1944.
86. Story, R. D. and U. Sagild. *J.A.M.A.* 152: 312, 1953.
87. Scrimshaw, N. S. *et al. Fed. Proc.* 15: 977, 1956.
88. Venkatchalam, P. S. and V. N. Patwardhan. *Proc. Roy. Soc. Trop. Med. Hyg* 47: 169, 1953.
89. James, W. H. and ElGindi. *J. Nutrition* 51: 108, 1953.
90. Medical Research Council. *Vitamin A Requirements of Human Adults.* N.R.C. Spec. Rep. Series, No. 264. London: H. M. Stat. Off., 1949.
91. Altschule, M. D. *Arch. Path.* 38: 71, 1935.
92. Bernhard, K., G. Ritzel and K. U. Steiner. *Helvet. Chim. acta*, 37: 306, 1954.
93. Sobel, A. E., L. Besman and B. Kramer. *Am. J. Dis. Child.* 68: 257, 1949.
94. Jonxis, J. H. P. *Acta paediat.* 36: 77, 1948.
95. Guilbert, H. R. and G. R. Hart. *J. Nutrition* 8: 25, 1934.
96. Guilbert, H. R. and W. R. Hinshaw. *J. Nutrition* 8: 45, 1934.
97. Clayton, C. and C. A. Baumann. *J. Nutrition* 27: 155, 1944.
98. Haig, C. and A. J. Patek, Jr. *J. Clin. Invest.* 21: 309, 1942.
99. Booth, V. H. *J. Nutrition* 46: 13, 1952.
100. Crabtree, C. *Anat. Rec.* 79: 395,1941.
101. Wald, G. and P. K. Brown. *J. Gen. Physiol.* 35: 797, 1952.
102. Bern, R. A., J. J. Elias, P. B. Pickett, T. R. Powers and M. N. Harkness. *Am. J. Anat.* 96: 419, 1955.
103. Flesch, P. *J. Invest. Dermat.* 21: 421, 1953.
104. Redfern, E. R. and D. H. Strangeways. *J. Anat.* 91: 391, 1957.
105. Popper, H. *Arch. Path.* 31: 766, 1941.

106. Lowe, J. S., R. A. Morton and R. G. Harrison. *Nature, London* 172: 716, 1953.
107. Festenstein, G. N., F. W. Heaton, J. S. Lowe and R. A. Morton. *Biochem. J.* 59: 558, 1955.
108. Lowe, J. S., R. A. Morton, R. G. Harrison, N. F. Cunningham and J. Vernon. *Biochem. J.* 67: 215, 1957.
109. Fell, H. B. and E. Mellanby. *J. Physiol.* 119: 470, 1953.
110. Mason, K. E. and E. T. Ellison. *J. Nutrition* 9: 735, 1935.
111. Wolbach, S. B. *Science* 86: 509, 1937.
112. Roger, F. C. and J. A. C. Brown. *Trans. Roy. Soc. Trop. Med. Hyg.* 51: 271, 1957.
113. Blumenthal, C. J. *S. African M. J.* 24: 191, 1950.
114. Waterlow, J. C. and S. J. Patrick. *Ann. New York Acad. Sc.* 57: 750, 1954.
115. Arroyave, G. *Rev. del Col. Med.* 7: 228, 1956

RESEARCH AND TRAINING IN NUTRITION

Introduction

KINNEY: The present conference came into being as the result of concern on the part of the Pathology Study Section of the United States Public Health Service regarding the progress and future of pathology in this country. For the past several years the Pathology Study Section has been systematically exploring the needs in research in pathology and whenever it has found an area of study or interest which appeared to be neglected, it has tried to support such an area in a practical way. One such area was diseases of nutrition. Consequently, a subcommittee of the Study Section was appointed to inquire into ways in which this area could be aided.

The Subcommittee suggested that a conference be organized which had as its purpose two basic objectives. The first was to assess the present status of training opportunities for individuals who were interested in nutritional research. The second objective was to determine those areas in the field of nutrition which were in particular need of development. The Pathology Study Section approved the suggestion and the National Heart Institute was kind enough to approve the Study Section's request for funds so that the present conference could be held.

It was hoped that such a conference would define the important problems in nutrition and point up deficiencies in our knowledge, as well as indicate areas where there is need for further investigation. It was thought that such a conference would aid in establishing the promotion of meaningful communication between biochemists, nutritionists, pathologists and other scientists interested in nutrition. This, in turn, might bring about a widespread realization that the continued isolation of the several branches of science interested in nutrition is a serious obstacle to progress in this field.

This conference was to be asked to review the present training opportunities throughout this and other countries. If, as the committee believes, there are certain inadequacies in the opportunities for training and in facilities for such training as well as in research in nutrition, then it was expected that the members of the conference would offer valuable suggestions for correction of this situation.

The planning committee selected the three diseases which we have been discussing for the past three days. Now we should indicate important research that is needed to fill in the gaps in our knowledge of these diseases. I suspect that many of the suggestions which will be made regarding research needs for these three diseases will have their counterparts in other deficiency diseases. Having discussed the more specific needs for research in these diseases, we shall consider the more general problems of training and research.

Suggested Needs For Research

KINNEY: In order to give form and direction to our discussion, groups made up of the chairman, secretary and the member who discussed the pathogenesis of each of the three diseases were asked to summarize the important needs for research for that particular disease. This will be followed by open discussion and I hope each member of the conference will participate freely.

Beriberi

The group for beriberi was made up of Follis, Burgess, Higginson and Mackenzie. Follis, will you please open the discussion?

FOLLIS: The group is impressed with the seriousness of the problem of infantile beriberi, a disease characterized by its appearance in breast-fed infants of a particular age who die acutely. Its relation to

thiamine deficiency has been brought out by therapeutic cures. The most immediate problem is to define more precisely its prevalence in terms of maternal nutritional status, the nutritive properties of breast milk and other environmental factors. Hence, field studies are necessary to gather vital statistics relative to infant mortality rates, feeding habits, mode or cause of death, etc. Even more important needs are clinical and laboratory tests in the mother and infant so as to define the plane of malnutrition upon which this disease occurs. Such would include physical examinations, correlated with chemical determinations on blood, milk and possibly urine. Studies of other pertinent nutrients, as well as the presence of factors such as anemia, infections, etc., are important. The minimal clinical criteria for diagnosis, with particular emphasis on the reliability of neurological findings, need to be delineated, as well as to determine which laboratory tests are the most pertinent. Such studies must be carried out by trained clinicians, particularly pediatricians, as well as biochemists and epidemiologists.

Similar studies must be carried out in the clinic in areas where the disease is prevalent, in pregnant and parturient women and in their newborns. Here it may be advisable to assess the excretion of certain nutrients, particularly thiamine, in terms of their intake so as to arrive at correlations of blood with milk, milk with infant serum, etc.

No modern functional or anatomic pathological studies are available which deal with infantile cases of beriberi or of the disease in adults. We are concerned by the complete lack of any current information relative to the pathologic anatomy of the heart or nervous system.

While preventive measures are of paramount importance, we must stress the continuing need to understand the pathogenesis of beriberi, for if we are to treat disease intelligently, we must know as much about its basic mechanisms as possible.

The above deals with the problems of the field. It is obvious that the program envisaged provides for the application of personnel trained in clinical and laboratory methods to carry out such studies. This, in a sense, brings the laboratory into the field. The opposite will be just as beneficial, that is, bringing field problems to the experimental laboratory for solution. Such studies should include attempts to reproduce a beriberi-like syndrome in various animal species. Data are still not sufficient to determine the exact role of thiamine in an experimental or in the natural beriberi syndrome. Cautious attempts might be made to study the experimental syndrome in human subjects, so as to give information as to the metabolism of thiamine and other essential nutrients on diets similar to those employed in the natural environment.

In summary, future progress in dealing with beriberi, particularly the infantile form, will be seriously hampered if more information concerning its prevalence, clinical course, laboratory diagnosis and pathologic physiology and anatomy is not forthcoming. A vigorous program which will provide an immediate liaison between field and laboratory is of utmost importance to fulfill this need.

In Central America and in many parts of Africa there are certain common forms of heart disease which do not conform to any of the generally accepted characteristics of heart disease. Such have been described under such terms as 'thrombotic surgical heart', 'endomyocardial fibrosis', 'cardiac collagenosis', 'ideopathic-African heart disease', 'cryptogenic heart disease', 'nutritional heart disease', 'chronic myocarditis', etc. In view of the possible relationships of such cardiac disease to endemic beriberi heart disease and chronic malnutrition, nutritional factors have been implicated in their etiology. The group, therefore, feels that this subject warrants considerable further investigation. Perhaps the first step should be to form a study group to share their information and to determine the extent of future study and research.

This conference appears to have brought out the need for close contact between field investigators and their coworkers in the laboratory. Let us hope that such cooperation may continue and grow stronger.

DARBY: The importance of understanding and research of the cultural patterns which are really the basis of the diseases which we are talking about should be emphasized. I believe that in our recognition of research

needs this aspect of the problem should not be neglected.

RAMALINGASWAMI: I want to emphasize the importance of a study of polyneuritis in pregnant and parturient women. Seemingly, there are many cases of polyneuritis in this group. Whether it is merely B_1 deficiency or something else, requires investigation.

I suggest that a little more emphasis be placed on the significance of accumulation of products of carbohydrate metabolism in determining the cellular pathology that one sees in beriberi. A pointed study of this relationship has yet to be brought out.

The assessment of the total picture of beriberi in a community is important. I had great difficulties in formulating criteria to be used in assessing the problem of beriberi in an area. This would involve a consideration of infant mortality rates, the study of patients in maternal and child health centers, biochemical estimations and other types of approach that could be used to assess beriberi in a community. We should recommend further study of the relative value of each of these types of approach for evaluating the problem.

For a field worker looking for signs and symptoms of beriberi in any stress group, examining the heart carefully is going to be extremely difficult. He has to confine himself mainly to a clinical study of the nervous system, which is all that is possible to do under the field conditions in which this disease occurs. It would be useful if the group could put out a statement as to what minimal neurological criteria one could use for the diagnosis of a neuropathy.

Endemic Goiter

KINNEY: The group for goiter consisted of Scrimshaw, Handler and Hazard. Will you begin the discussion please, Scrimshaw?

SCHRIMSHAW: Our committee has listed what they consider the most pressing needs for investigation in the field of endemic goiter. These are *1*) the production of a goiter experimentally which corresponds to human endemic goiter; *2*) the biochemical pathways of thyroid hormone production in endemic goiter; *3*) the problem of exogenous goitrogenic factors in areas of iodine-resistant goiter; *4*) to separate thyroid changes due to iodine deficiency from those due to other factors in an endemic area; *5*) genetic goiter areas; *6*) studies of the function of thyroxine and its action upon body tissue with special reference to *a*) its relation to the development and function of the central nervous system, with special attention to deaf-mutism and cretinism, and *b*) the mechanism by which it influences metabolic rate.

STANBURY: Please amplify the fourth point. I didn't quite grasp its implication.

SCRIMSHAW: The fourth point referred to the facts brought out in the discussion of the frequency with which changes due to iodine deficiency are described as characteristic of kwashiorkor or other conditions, and the intent of this point is to ask the pathologist to sort out those changes in thyroid glands in an area which are due to iodine deficiency from those which are due to other factors.

FITZGERALD: It should be remembered that this is an excellent area for the pathologist who is interested in trying to correlate function with morphology. He has some new techniques such as electron microscopy and radioautography which should aid him in getting some fundamental and important functional information.

RAMALINGASWAMI: The last of the six points made by Scrimshaw for study relates to the function of thyroxine. Scrimshaw suggests a study of the role of thyroxine in the central nervous system and basal metabolic rate. One might suggest study of other aspects too, such as the relation of thyroid to connective tissue, lipoprotein, etc.

SCRIMSHAW: The way this was worded wasn't intended to exclude other studies of function of thyroxine. It was felt that its role in the central nervous system was so basic to the whole production of cretinism, mental deficiency, and the other presumably grave consequences of iodine deficiency, that these were points which deserved special study.

MATOVINOVIC: Scrimshaw, did you mention the problem of iodine requirement, and the form of supplementation of iodine in terms of iodide and iodate? Because it is quite important.

SCHRIMSHAW: In regard to the failure to mention the need for more data on iodine requirements, certainly more information is

needed. This was recognized in the discussion, and by the group. Perhaps it should be a separate recommendation, but it was felt that the problem of iodine requirement in endemic areas hinged to a considerable extent on the operation of goitrogenic factors, and that from an understanding of the operation of goitrogenic factors in endemic areas would come a more realistic appraisal of levels of iodination required, and it was also intended to cover the need for more information on cretinism.

OOMEN: Beriberi is a problem which is not quite clearly defined and which is still very much in need of identification. The same is true for xerophthalmia and cretinism; perhaps it is less true for endemic goiter. This is very important because in the case of beriberi in the Philippines, the diagnosis is far too popular. Take xerophthalmia in certain parts of Indonesia and other parts of the world, this diagnosis is made often enough but the implications are not understood. So one of the real problems is still identification. Then I should very much like to come back to morphology, and to autopsies, to the ordinary means of a clinician to find out what the condition is, and to have accurate statistics, otherwise we have no firm soil under our feet to formulate problems.

SCRIMSHAW: Apart from formal commissions and committees established to determine definitions, the best way to straighten these things out is through personal interchange at meetings such as this and through the visits of persons from one laboratory to another. To the extent to which this conference in its recommendations can encourage this type of international interchange and, particularly, financial support for visits of people from one region to another, it will contribute to the matter of definition. Further, our conference will also contribute very greatly to this matter of knowledge of the literature, for one of the revelations of visiting another country or continent is learning about the extensive hitherto unknown literature in their medical journals.

Hypovitaminosis A

KINNEY: The group on hypovitaminosis A has for its members Aykroyd, Nadel and McLaren. I believe that Aykroyd will start the discussion on this topic.

AYKROYD: The material presented at the conference shows clearly that hypovitaminosis A is a serious problem of considerable magnitude in many parts of the world and is responsible for much preventable blindness. It also demonstrates that xerophthalmia and keratomalacia are usually diseases of the young child. A number of factors, such as infant feeding practices, maternal nutritional states with special reference to vitamin A, the lactation performance of the mothers and intercurrent infection and infestations, influence the epidemiological picture of hypovitaminosis A in a community. In some areas, such as the island of Java, there is evidence to indicate that concomitant deficiency of protein influences the metabolism of carotene and vitamin A. All these factors require further investigation.

It was agreed by the group that existing knowledge regarding vitamin A is extremely limited. It was felt that there was need for additional research in the following areas:

1) The development of methods for detecting and recording signs of vitamin A deficiency.

2) Studies to determine the incidence of vitamin A deficiency.

3) Determination of the basic requirements for vitamin A and carotene in the human. Further, there is need for a better understanding of the dietary, metabolic and pathologic factors which influence the absorption of carotene and its conversion into vitamin A.

4) Development of ways for the use of appropriate sources of vitamin A and carotene in prevention of deficiency. This would include the use of both concentrates and foods which are locally available.

5) The study of the pathology of vitamin A deficiency. Particular attention should be given to the relationship between changes due to vitamin A deficiency and those due to deficiency of other nutrients.

6) Basic physiological and biochemical research on the mode of action of vitamin A.

OOMEN: One of the most elementary things we need is a good registration of the cases of vitamin A deficiency and a classification and standardization of the nomenclatures, for it is a very important problem which

field workers everywhere in the tropics encounter every day.

MASON: It is appalling that we have had no significant new knowledge added to what we knew in 1935–40. For a disease which is so common in certain parts of the world, it is a shame there is such a lack of information on the full pathologic picture in various organs and tissues. This is something that cannot be performed in this country, but it should be encouraged to be done in areas such as Oomen is working in through adequate support to pathologists who have the material at hand.

DARBY: It might be important for us to recognize what we really mean by support, particularly to pathologists working under conditions where they have a tremendous demand on their time and very little in the way of support for investigative work. This bears directly on the need for additional trained personnel in environments where deficiency diseases occur, and while I'm sure that our encouraging the giving of money will be useful, it is not going to solve the problem of getting work done in the absence of pathologists to undertake the necessary investigative work.

SCRIMSHAW: Pathologists working in the areas where the type of material we are interested in are usually overworked. Often they would like to do research but they never quite get around to it. We have found that just a little financial help—$25 to $50 a month—will enable some individuals to hire a medical student to help with their routine work and to permit them to do some research. The availability of small sums of money to individual pathologists wanting to do research can be a practical means of doing something now.

BURGESS: Darby said earlier today that perhaps bringing a couple of anthropologists into this meeting would have been a good thing. Java is probably one of the greenest places in the world, and yet Oomen tells us there is a great deal of avitaminosis A. This disease could be stopped if people would eat the right foods. I suggest that we find out why they don't use the means that are there to prevent the disease now.

NADEL: This group could also consider field research of a different sort. For example, in the problem of hypovitaminosis A, it seems from McLaren's previous presentation that this is a vitamin which a population can store for a long time, and if there was a means of getting large doses of the vitamin into the population this would take care of, say, an immediate disaster-like problem in a localized area. Is there room for research to determine the best means of getting a needed nutrient to a people and its children in a short period of time?

SCRIMSHAW: This is an expensive and complex thing and it requires anthropological assistance. It doesn't involve going into one area and trying one thing but, rather, you have to try a number of different things under a number of different circumstances. Ordinarily it can't be done in a hurry because you get an initial acceptance of a program which is not an indication of the effectiveness of the program over any reasonable period of time. While this kind of thing needs to be done, its difficulty and its cost shouldn't be underestimated.

PLATT: I have had bitter experiences with this kind of problem, and I believe that we should recognize that the block in all of this work is at a very high level. You've got to get responsible statesmen, ministers and people interested in this sort of a problem. One of the possible fruits of this meeting is an understanding that these diseases require not only basic research but a recognition of these other problems which must get to the ears of the people who can say, "If we've got these things, what are we going to do about it?" I'm sure that we mustn't forget that there is this block due to lack of interest, perhaps, or ignorance on the part of very senior and responsible people.

SCRIMSHAW: May I make a general comment which more or less applies to all three sections? That is, that until fairly recently there has been a tendency to pay attention to individual diseases. When nutritional diseases were studied by nutritional surveys, the emphasis was upon nutritional diseases and the surveys did not pay much attention to infectious diseases. Further, the persons concerned with infectious diseases generally weren't concerned with malnutrition. In the discussions these last three days it has been apparent that there is an increasing realization of the interrelationship between nutritional and infectious diseases. It has been

clear from other studies not discussed here, that the synergism between infectious diarrhea and protein malnutrition in the production of kwashiorkor, for example, is very close. It has been suggested that both enteric diseases and systemic infections influence beriberi. Our report should reflect this relationship and the need to understand the extent to which both enteric and systemic infections in technically underdeveloped areas operate as stress factors to precipitate nutritional deficiencies and to cause even death where neither would occur without the stress factors.

Training and Research

KINNEY: Darby was asked to serve as chairman of a group to organize the discussion regarding training and general research needs in nutrition. Patwardhan, Ramalingaswami, Stare, Moon, Olson and Kinney worked with him. It is planned to have Darby begin the discussion and the others speak on certain topics which are of particular interest to them.

DARBY: This group raised three rather basic questions. First, what is meant by nutritional pathology? Second, how are personnel attracted into the area of nutritional pathology? Third, what opportunities are needed for training and research?

Relative to the question of the meaning of the term, nutritional pathology, it was agreed that this is really a study of nutritional disease in the broadest sense, including deficiency disease, disease of nutritional excess, those derangements in nutrition which occur in metabolic disorders and the nutritional components of various other conditions, such as infectious diseases. The consensus was that we're not concerned simply with tissue pathology but with the many aspects of the pathophysiology of disease, and to understanding these many aspects in such manner as may contribute to our knowledge of the pathogenesis of disease as seen in man. Four categories or types of study were recognized, i.e. a study in what we might call the defined field of pathology; a clinical study of disease; a laboratory study of disease; and field studies.

As to the matter of training, it's clear if one looks at nutritional pathology from this rather broad view that the training experience of the individual must include both experimental and observational approaches. The individual must have an opportunity to become acquainted with the rather broad concept of disease, man versus environment, which was so well illustrated by Follis' discussion in summarizing beriberi.

One of the things we have to do in recruiting personnel is get their attention. There were several ways suggested to do this. One was that means be found to bring about early exposure of the individual to the subject of nutritional pathology. Some of you are aware of this technique. Scrimshaw is, because each year he has a number of medical students from the United States who work at INCAP and also has a number of medical students from Guatemala working with him. A number of these people have indicated quite clearly that they are continuing in the field of nutritional pathology under the broad term that we have discussed. So that early exposure and contact is important.

It also was noted in our discussion that interest limited to nutrition is not sufficient to attract people to the area of nutritional pathology. This nutritional interest really should be an intricate part of the professional structure of the individual. So that our group was not in favor of trying to set up a lot of separate nutritional scientists, but rather have broadly trained scientists realize that nutrition is a legitimate part of biochemistry, of pathology, of clinical medicine or epidemiology, or whatever the field may be. If one has adopted this approach it's obvious that he cannot limit the trainee experience too rigidly. Hence, in preparing trainees one must provide for an adequate breadth of experience through opportunities to become proficient in general medicine, pathology, and the laboratory approach to research with experience in areas where nutritional disease occurs.

Another need is to provide scientific personnel, at all stages of development, opportunities for training and research in suitable

and stimulating environments. It was indicated that if we are going to profit from exchange of personnel, whether that person is coming from another country to the United States or going from the United States to another country, the individual should remain for a minimum of a year.

It was strongly urged that opportunities for support of individuals at different levels of experience be made available, that is, for young trainees and for established individuals as well. It was felt that such an exchange would go far toward correcting the often held misconception that the opportunities for training provided in a more sophisticated environment are necessarily better. It would also remove the unfortunate temptation of young, inexperienced trainees to transplant their investigative activities from one region to another without really considering the opportunities which exist in their own environment.

It was apparent that much of the needed information in nutritional pathology and understanding of human deficiency disease might be obtained in the environments where these diseases occur. Hence, the strengthening of training and research institutions in those areas is essential. Only by doing so can sufficient personnel be developed to provide the information such as we have asked for as a result of our three days of deliberations. We did discuss the kinds of institutions where training might be obtained; of course, in universities and medical schools one expects such training. However, there are institutes of various kinds around the world where such training experience may be obtained. There are so-called research stations, both of the temporary and permanent type, where such training is available or could be amplified. These laboratories are either supported by or often identified with either governmental or international groups or agencies. I think the first priority would be given to the strengthening of established facilities, but not to the exclusion of the establishment of new facilities. It was emphasized by the group that facilities are secondary to individuals and that the support of the individual is the most important contribution which can be made to the development of the best training facilities.

Finally, our group recommends that there be additional support directed toward providing for purposeful movement of individuals from the field to the laboratory and from the laboratory to the field, regardless of where the field opportunity and the laboratory opportunity may be located. We would be negligent if we didn't underscore 'purposeful', because we do not wish to endorse 'junkets' in contrast to the profitable exchange of personnel.

TEJADA: I would like to comment about training and research in the nutritional field, particularly geographical pathology in relation to nutritional disease. One of the worst handicaps that pathologists all over the world have is the lack of communication between each other as far as nutritional pathology is concerned. Sometimes we are interested in seeing how a heart from Africa looks and we want to compare it with a heart from Latin America and a heart from India, but we don't have this type of correlation. I consider that the entire world is really a kind of laboratory, a natural laboratory, and each country is really an experiment. If we can study the experiments, the natural experiments in each country, later on we can find what are the differences between one country and another. For instance. we are studying atheroslcerosis in Guatemala because it is said that we don't have atherosclerosis in Guatemala. That's something important to know. What is the difference, for instance, in the incidence of atherosclerosis in the United States and Guatemala? This is a natural experiment. If we can establish the basis and we can definitely show that there is a difference, then later on we can go back and establish what the differences are between one place and the other. I could give you many other examples. These types of experiment are really what I consider to be the future of the pathology of nutritional disease. So I suggest that this conference ask an international organization to establish some centers to aid in the study of the pathology, and to have relations through correspondence as well as a code to allow us to work on a common basis. We need to have some regional centers in each continent, and different field units in each country of each continent. Then we shall have a real basis for comparison of

our natural experiments that are occurring throughout the world.

KINNEY: In other words, you are thinking in terms of a clearing house for tissue to which you would ask men from different countries to agree to become participating members. They would then undertake to supply the various types of material needed for controlled experiments, or for the sake of comparison. Such an arrangement should not be too expensive to set up. Tissue would not be stored at such centers. What would be needed at the regional center would be a knowledgeable individual who could obtain material when requested.

HIGGINSON: The whole problem will be one of personnel. You can't superimpose a nutritional pathology service on an inadequate service now in existence. They are people who are considerably overworked running a service unit. It is unsatisfactory to plunk down somebody with special interests in the same laboratory. It only makes for friction. So you have to start by strengthening the basic pathological services. Whatever suggestions that are made in regard to research and training should always take into consideration the framework that is present. Further, there is no substitute for an enthusiastic local worker on the spot. If you haven't that you can wash out any attempt to produce work on the pathologic level in that area.

SCRIMSHAW: This matter of exchange of material is helped immediately when people from one area spend some weeks or months in another. Through the very active fellowship visit program of WHO, we have largely resolve the regional controversies over clinical science and basic features of kwashiorkor relative to caloric deficiency and so on. Because particular attention has been given to this, there has been some degree of interchange between almost all major centers interested in kwashiorkor in the world. Secondly, the exchange of pathologic material and information on disease can be facilitated by making funds available for these more prolonged exchange visits.

RAMALINGASWAMI: Darby mentioned training, or creating an atmosphere at the undergraduate level. One would hesitate to add anything to an already overburdened undergraduate curriculum, but our experience in India is that in spite of so much malnutrition, very little time is spent on the teaching of nutrition in the medical schools. Through our Nutrition Advisory Committee in India we have been trying to get the medical schools to include more and more physiology and pathology of nutritional disease in the medical curriculum. I found in the last three months that in teaching general pathology to the students, the use of references and illustrations in nutritional pathology and one or two laboratory exercises has had a remarkable effect on the students. I believe that if a little more attention could be paid to the permeation of nutritional knowledge at different points in the teaching of the medical student, in anatomy, physiology, biochemistry, pathology and clinical disease, some potential research workers might become interested in nutritional pathology.

The second point is that a man has to have a certain amount of contact with routine hospital pathology and contact with human disease existing in that area, and then has to have a special technique which he uses in the study of any particular problem, as, for example, electromicroscopy, histochemistry, cytopathology and so on. In this connection, there is a one-year training course given in Oxford, which is known as the honor school in physiology, at the end of the preclinical training. That is, after one and one-half years of anatomy, physiology and biochemistry, a medical student may spend the year doing advanced biochemistry and physiology. One can describe this course as learning the grammar of medicine. If some of the best students in the medical schools can be selected and put through a course like this, I have a feeling that one would get workers who would be much better qualified and suited to study nutritional disease.

The third point that came up was that nutritional pathology should not be a specialized discipline by itself but should merge and permeate the various disciplines of the medical school, so that we get more and more people with broader training to look at nutritional problems rather than a person who had specialized in one particular aspect only.

Also, I wonder if it will not help us if some arrangement could be made with the universities by which a person going out and

spending some time working at a problem, say in India or Thailand, could utilize the work that he does there for a thesis for a higher degree. I am really thinking of our own Indian universities. A person from my department could spend some time in Burma or in Ceylon, or wherever there is a problem, and receive credit for the work that he does there provided due consideration is given to the standard of the work and the quality of the supervision that he receives. If we could recommend to whatever organization we have in each country, national medical council or universities, that they provide this kind of facility for the attainment of higher degrees for work done in places outside the university which gives the degree, it will help in attracting some young people to take up investigation of problems. We have had a few young men who want to do investigative work and yet if they join an institute which is not recognized by the university or have to work in the field, that work is not recognized by a university and they hesitate to take up such a career.

MOON: I'd like to speak first on the need for recruitment of personnel. It is necessary to recruit at all levels and we should not be put in the position of robbing Peter to pay Paul because of the shortage of scientific personnel in various specialities. The question comes up, what is the best mechanism to utilize? Are there several mechanisms that can be utilized to increase the research potential in this area? I would agree with the previous speakers who have mentioned the fact that it is necessary to begin recruiting or interesting people at an early age. In this regard it might be very desirable to strengthen and encourage the existing programs of education in colleges and in the professional schools in the principles of nutrition. I think that at least as far as our school is concerned, nutrition is not taught as adequately as might be desirable. Looking at the situation realistically, I think that even if such a program of recruitment were instituted tomorrow, we should not expect a significant increase in personnel for 5 to 15 years. And I think that this should emphasize the fact that positive action is desirable at the earliest possible date.

This, then, brings up the question of training centers. It seems to me that there must be a good many centers that are available for the training of personnel, yet unless we have adequate information as to where these centers are we would not be in a position to make any recommendations concerning establishment or strengthening of centers. For this reason, I think that a survey, or at least some method of attaining this information, is necessary. Another point in connection with training centers is that it would be necessary also to explore possible means of support. As far as research is concerned, the various categories of need have already been mentioned. I simply wish to mention them again for emphasis. Firstly, the problems concerned with undernutrition including specific nutritional deficiencies. In the second category are the problems with which we are primarily faced in the United States—the problem of overnutrition and the problems of dysnutrition, that is imbalanced mechanisms. I think that in this last category is where we may expect major progress in the future. Finally, the development of newer and more sensitive techniques are necessary for progress.

STARE: I think the problem of training and research is very simple. It simply takes 'M and M' to have good training and good research. 'M and M' stands for men and money. It takes men that are well trained to do the work with ideas, which in most cases will come to them from out of the blue and not from their teacher. Because human beings are what they are, if their teacher had the idea he would have worked on it himself. It takes money to pay the men so they can work in comfort for their families and self—money to visit India and Indonesia and be stimulated by the nutritional problems they have there.

Those of us who are connected with any medical school or school of public health or graduate school in the United States know that in the last few years there's been talk, talk, talk on changes in education. I doubt if there's a medical school in this country which in the last few years hasn't instituted what to them is some very marked change in the teaching of medical students. This applies as well to schools of public health and graduate schools. So there is a lot of interest in education, and changes in medical education. But when we come to nutrition,

whether it is nutritional pathology, clinical nutrition, experimental nutrition, or any aspect of nutrition in our professional schools, the main thing we need is a man. It's nice if there are two men—but at least one man is needed to push the idea of nutrition in his particular school. It doesn't mean that he should do most of the teaching in nutrition, but rather he should seek out colleagues, whether they be in obstetrics or pathology, and try to stimulate them to do a little work in nutrition. The best medical school that teaches nutrition does not even give a course in nutrition, for nutrition is too broad a subject. Sixteen or seventeen years ago, I wrote on the teaching of nutrition in medical schools. This report gave a list of something like 72 concepts or subjects in nutrition that ought to be integrated at some place in the medical curriculum.

As far as stimulating people, obviously it is best if you can interest students when they're young. I can give you a few specific examples. One of our principal colleagues, in our department now and in charge of most of our monkey work in atherosclerosis, worked in our department off and on during his third year and fourth year in medical school and then stayed on with us. But it is not necessary to start them young, and I can give you examples of individuals who started older and who were stimulated in part through contact with people in our department.

In summary, I would simply say that I am not much in favor of institutes. It takes men and money. Now unfortunately or, perhaps, fortunately, men and money seem to develop, if there's enough, into institutes. We're hoping that the younger people can be provided with an adequate salary, will have facilities to work, and will be able to see things in other parts of the world. The keynote to the teaching of nutrition in any professional school that involves health is integration.

PLATT: We get a number of people from overseas, and it's desirable that not only should they be trained but they should have some evidence of it. Therefore, we're experimenting with a master's degree which takes 2 years. In the first year, the student studies either physiology or biochemistry or both. In the second year, he tackles a research problem. He then becomes a graduate of the University of London. We hope this will encourage students when they go back to work for an external Ph.D. which they can do as graduates of the University of London. They can work on their own problems in the field or under supervision and submit a thesis in 3 years' time for their doctor's degree. We have had some success in that there are people about the world now who have been with us and are now devoted research workers in nutrition.

OLSON: I was asked to comment briefly on the clinical and laboratory aspects of training and research. I thought I might tell you something about the program which we have envisioned in our institution for training clinical nutritionists. At the present time, we are dependent upon the process of spontaneous attraction of physicians and biochemists to the field of nutrition. This has been going on in established centers which have not been organized primarily for training but have been organized to do research. Able people have been attracted into their research organizations and, in the process, been trained and gone out to form nuclei in different institutions to promote nutrition. However, the present need for both pathologists interested in nutrition and clinical nutritionists is so large that some more organized attempt must be made to organize centers and to develop programs which will produce individuals who are qualified to become additional nuclei to promote clinical nutrition and the pathology thereof.

We've been appalled with the disinterest and ignorance of the average physician in nutrition. The subject of nutrition has been ignored in our medical schools even more than in similar institutions over the world. The dominant influences in the field of nutrition in this country are nonmedical. The emphasis should be on the physician. With this in mind we have thought about starting a program for students with a good undergraduate training who have had an internship and, hopefully, a 1-year residency in medicine. These individuals would go through a 3-year postgraduate program in which the first year would be devoted essentially to basic science, biochemistry, physiology, statistics and other allied medial sciences bearing on the science of nutrition.

The second year would be spent in a review of clinical medicine in terms of nutritional illness as it is seen in this country. This would be largely in the area of over-nutrition, but to a limited extent in the area of under-nutrition which we see in alcoholics. It would re-emphasize the fact that a general evaluation of health must be done, with an inventory taken of possible communicable disease, parasitism, other chronic degenerative ailments as well as nutrition, fortified by an adequate medical and dietary history, a physical examination and appropriate laboratory tests. This would be designed to place illness in the proper perspective in the evaluation of the health of a patient. The third year would be assigned to a research problem carried on either in this country or possibly abroad. The exchange of people over the world to see first hand both under- and over-nutrition would improve the clinical judgement of the individual. We visualize that this program could lead to an advanced degree but it need not. We hope that such a training program might provide men who are qualified to take posts in universities with some stature so they could argue from a position of strength to influence curriculum and research in the field of nutrition.

PATWARDHAN: While countries like the U.S.A. have admirable facilities and opportunities for studies of metabolic disorders of over-nutrition and imbalances in nutrients, the actual problem of the study of deficiency disease can best be done in the region where these diseases occur. One of the principal recommendations of this conference should be an evaluation of research facilities which exist in these regions, and, if possible, to take steps to strengthen them. Various solutions have been suggested. Facilities could be strengthened by increasing the number of personnel interested in nutritional research and by adding additional equipment. Further, opportunity could be given in these centers for interested pathologists from countries like the U.S.A. to enlarge their experience and study the disease at first hand. While you may change the conditions for experimental work, the actual interrelationship and interplay of various factors—the dietary, cultural and environmental—in pathogenesis of nutritional or deficiency disease can best be learned by work on the spot. I would, therefore, suggest that institutions which already exist in these regions should provide facilities for workers from countries outside the region to work for at least a minimum period of 1 year, so as to get the full impact of the problem which they are going to study. This will help them after they return to their country to carry on experimental work with full knowledge of what actually occurs when deficiency disease appears in these regions. Otherwise, most of the experimental work loses touch with reality, and often leads to unsatisfactory conclusions with regard to pathogenesis. These institutions have to be identified, as you say in this country, and for the purpose of enabling your people to come and work in these centers, funds also will have to be provided.

We would also like it if some of the people who work in our laboratories could be afforded facilities to visit the U.S.A. The object would be to learn newer techniques to handle equipment which we can buy but sometimes cannot use because of lack of specialized knowledge, and thus increase the potential of our research work in our countries when these people return. Money will have to be found for this either by the countries concerned or by the richer countries.

SCRIMSHAW: Another aspect of training which has been only briefly mentioned is the training of public health workers in the field of nutrition. One of the greatest difficulties which Burgess and I encountered in attempting to develop nutritional activities within the World Health Organization and the Pan American Sanitary Bureau is that the health specialists themselves have not been taught in the schools of public health that nutrition is a part of their job. We are continually in the position of trying to impose something upon them which, while recognizing its importance, they don't see quite where it fits in with routine public health activities. This goes back to the failure to teach nutrition as part of the public health program.

As Stare has pointed out for the teaching of nutrition in medical school, it perhaps is an error to think that this requires a department of nutrition in every school of public health. It does require that the people teaching

public health practice, maternal and child health, epidemiology, environmental sanitation and the other specialities bring in nutrition where it is involved in their disciplines and act and talk as if it were important. I'm not sure that there are any schools of public health which require the candidates for a master of public health degree to study nutrition. There are optional courses, but until it's impossible to get a master of public health degree without acquiring some adequate background in the principles of nutrition, our efforts within the public health organizations, and our efforts with the health officials in the individual countries, under whom most of the nutrition work falls, will continue to present sometimes as much difficulty as the nutrition problems themselves. In recommending training and teaching programs, special recognition should be given to the necessity of incorporating nutrition principles in the training of every type of public health worker.

FITZGERALD: In terms of recruiting residents in pathology, although this would apply to any other discipline, one of the best ways to recruit people into a field is to provide fellowships specifically labeled. The great successes of the American Cancer Society and the cancer trainee program set up by the USPHS are good examples, and it would pay to specifically label fellowships in nutritional disease, particularly at the residency level.

SCHAEFER: I would like to mention the program of the Interdepartmental Committee of Nutrition for National Defense because we have had an opportunity of tackling some of these problems. The number one problem is the recruitment of people. During the past 2 years, we've conducted 8 surveys. In summary, this means that we have performed 50,000 clinical examinations of individuals throughout the world, 5,000 blood and urine analyses, as well as dietary surveys, and a real consideration of the sociology, anthropology, food availability, agriculture and economic potential of these countries.

In the first survey, it was difficult to get clinicians, biochemists and others interested in going on just a 3-months' tour, let alone a year's tour. As the program developed we have been able to get new personnel, and whereas before we had to literally beat the bushes for a man to join the team, we now have a waiting list of people who've gone back to their universities. I don't know whether we can call them inspired, but at least they become interested. They have become appreciative of problems that don't exist in this country, and are interested in world nutrition. Therefore, we have been able to stimulate interest in nutrition in 23 universities throughout the United States.

There are tremendous opportunities for following up this work in the eight areas where we have been. We have ignored study of any specific tissues in this initial survey. But the opportunity exists, and the mechanism has been set up where, with very little effort, we could get people from our universities over there to work with their counterparts. This is not new to the technique that's used in FAO and WHO, but I think it has helped stimulate and reactivate interest in nutrition and affords at least a start for this exchange of personnel from the developing countries with the United States.

HANDLER: I worry when you talk about training a nutritionist. I don't know what a nutritionist is. I seriously doubt that training anyone as a nutritionist will solve any of the problems which we have discussed at this conference. You can produce technicians, but you can't train scientists as nutritionists in the sense in which the term is being used. And if any of these problems, which have cried for answers in the last several days, are to have answers, you will have to obtain them from people who have been trained as scientists. This means going back to people who are very thoroughly trained in the disciplines which we have recognized for a long time. If some of these disciplines have not caught up with the times, then it is for the people in these disciplines to do something about it. I think to some extent this is what caused this conference to come into being. Pathologists themselves have been concerned with the training of people whom they were thinking of as experimental pathologists. But I warn you against setting up institutes for training nutritionists. The people you need will think of themselves in other senses, as chemists, as physiologists, as pathologists, as

clinicians. They must be as thoroughly trained as possible in these disciplines. Then you can use fellowships or use anything you will to entice people into investigating problems in the area in which you are concerned. If you're going to train people as nutritionists, rather than in one of the fundamental disciplines, I think you will be doing the cause of nutritional research a great disservice.

MACKENZIE: I agree with Handler. We need people rigorously trained as investigators, and the last thing we should call them is 'nutritionists,' because to most of us it means 'home economists.' Nutritional problems are known. What we need are people to help solve them in the field.

OLSON: I disagree with what Handler and Mackenzie have just said, for this reason: it's been customary in medicine to train people for specialities. This is not to say that every person who is trained in cardiology becomes an outstanding heart specialist. But these men perform a useful function in the community in the practice of medicine. And it is in some such manner that I think a program should be designed to equip physicians to study and understand nutritional disease. This is not intended to produce mature investigators who are going to go to Indonesia and solve the vitamin A problem. This is certainly not the intent, but there has been a lack of emphasis upon training physicians in the elements of clinical nutrition.

DARBY: I strongly suggest that the conference recognize that the nutritional interest of a scientist should be an integral part of his professional competence in a defined area, and hence, not a separate discipline. One cannot limit the trainee's experience rigidly, but rather it is essential that we provide for adequate breadth of experience of trainees through provision of opportunities to become proficient in general medicine, pathology, in laboratory approach to research and in field experience in areas where severe nutritional diseases occur.

KINNEY: I hope the conference can come up with some recommendations which might implement some of the suggestions that were made this morning. I think the best way to do this would be to ask Aykroyd and Burgess to tell us about their respective organizations and what they have done and are doing so that we will be in a better position to make suggestions.

AYKROYD: As far as FAO is concerned, we fully realize that we need research to support our practical program in nutrition and other fields. The United Nations agencies have not hitherto had funds for research. The announcement at the WHO assembly last week to the effect that substantial funds might be made available to WHO is definitely an innovation. Occasionally and in rare circumstances, FAO has supported research. We have carried out surveys in the field of nutrition through consultants and through our own staff which might be called research, but in general the amount of laboratory research which we have sponsored is small. We have a method by which both FAO and WHO are able to get needed research done without themselves becoming directly involved. An example is the committee on protein malnutrition of the National Research Council in this country which administers a grant for work on protein-rich foods, the money having been provided by The Rockefeller Foundation. The work done under that grant is closely related to the FAO-WHO-UNICEF practical program in the field of protein-rich foods and their development and use.

It is clear that a symposium of this nature, which brings together field workers from various regions in the world and research workers in countries such as the U.S.A. who have considerable facilities for scientific research, is in itself extremely useful. I think it is clear, when one considers this meeting and its course, that the voluntary workers have got a number of ideas from the people in direct contact with these diseases, ideas which they may find very helpful in designing their own research projects. On the other hand, the people in the field, whose concern is primarily with practical programs and projects, benefited equally from their contacts with people who are more specialized in scientific research.

With regard to actual types of research which might be supported by such an institution as the National Institutes of Health, it would seem to me that out of our discussions here a very considerable number of proposals and ideas for lines of research

have emerged which are of interest and of practical importance, and that there will be no difficulty in finding in the report of the symposium useful possibilities for research which might be supported.

BURGESS: The nutrition activities of WHO until relatively recently have not been very great. There has been a very small staff, and there has been a failure on the part of public health organizations generally to recognize the importance of deficiency disease. However, at the present time, there's a good deal more interest in nutrition, and I understand that at the meeting at Minnesota the Director General made reference to nutrition as one of the most important problens in future activities. WHO's activities in the past have been to define the deficiency problem. The prevention comes more into the field of FAO. I think it is unnecessary for me to say that this meeting, from our point of view, has been an extremely valuable one because we've been essentially discussing means of defining the problems.

Our research activities have never been very extensive. However, we have been able to make small sums available for research, but in general, research is not regarded as part of WHO's activities. We do, in the same way as FAO, give services to countries. We send people out to investigate disease conditions, and there are a number of people around this table who have worked for WHO in that particular field of work. Now the only other thing we have been able to do is to facilitate an exchange of workers, which is perhaps the most valuable thing we have done although the total sum of money involved has not been very great. We also run things like study groups which make particular studies of specific problems, but, on the whole, our activities at the present time are somewhat limited.

DARBY: It might be useful to consider some of the ways by which the National Institutes of Health might support research in these areas. One would be to extend the program of foreign fellowships, which currently is limited to fellows from Europe, to support fellows from other countries. This might possibly be restricted, not because one wishes to restrict it, but because practically there would have to be some limit to fellows working in institutions which have fellowship support or training grant support from the NIH. This might be one way of choosing a pilot group to try the efficacy of this type of support.

Another possibility is the support of exchange of personnel between two laboratories. This might be a very useful means of providing and encouraging scientific intercourse.

Another concept is support for working conferences in an area where there is endemic disease. It would be particularly valuable if half a dozen persons could get together and really work in an endemic area, and attempt to arrive at definite decisions about some particular disease. I can see a number of benefits from this type of support, because not only would it help get information and settle certain points, it would also provide a tremendous stimulus for research if a group of experts worked in a place for a period of from 6 weeks to 2 months. Also, it would provide a tremendous stimulus for work on a subject in a medical school or medical institution.

From time to time there will be further need for the holding of such discussions as WHO and FAO have engaged in under the Macy Foundation's sponsorship and some of their other joint studies, or joint study groups. Here again is an area in which NIH funds might be made directly available for help in such work.

KINNEY: Darby has given us three basic ideas to work on: one, a fellowship program; two, an exchange program; and three, the idea of working conferences. Not in any way to restrict the discussion, I suggest it would be more orderly if we first take up the idea of fellowship programs with the plan of either making or not making a recommendation regarding the matter of fellowships.

AYKROYD: I question if there is a tremendous need for further development of a fellowship program. We have our fellowship program and so has WHO, and there are half a dozen other bodies which have fellowship programs. I would like to put the question to Patwardhan, taking India as an example. Is it not perhaps true to say that as far as India is concerned, almost any able young man in the field of scientific research in nutrition who has wanted to have a fellowship there has found such help available from

some source or another? There has not been any general shortage of actual money for fellowships. Now it may be some improvements could be introduced into the fellowship program of the people who come from, let's say Asia to Northern Europe and America, or we might have this idea of fellowships for persons going from this country to work in other parts of the world, which I think is an excellent one.

PATWARDHAN: What Aykroyd said is largely true. In the past 10 years the FAO, WHO, Colombo Plan and DCM administration of the United States, as it was known then, supported a large number of young workers in various countries with fellowships. The support by the Colombo Plan and the DCM Administration cannot be compared with the fellowships granted by FAO and WHO. As far as my information goes the DCM and Colombo Plan fellowships were for longer periods. It means that a person could come here and stay for from 1 to 2 years for training. Quite a number of my own colleagues have been benefited by this procedure. On the other hand, FAO and WHO fellowships are for comparatively short periods, usually of 6 to 9 months. This period is ordinarily not enough for those who really want to learn or to do something worthwhile. These short-term fellowships have helped public health workers who are interested in food technology or in certain applied aspects of nutrition, either from the FAO point of view or that of WHO. However, if we are considering providing opportunities for young men from these places to come and work in well known institutions, with well known persons, then either the FAO and WHO programs for fellowships will have to be reoriented or funds will have to be found from other sources.

SCRIMSHAW: I'd like to strongly support what Patwardhan has just said, and indicate the situation in Latin America. There are fellowships available through the Pan American Sanitary Bureau, WHO, Kellogg Foundation, and so on, for people who want relatively brief training in public health. There are fellowships for someone to come up here for a year in connection with medical specialities for the improvement of medical schools, but there is a great scarcity of fellowships for training in the basic sciences, leading to a Master's degree in biochemistry or physiology or one of the fields which is not directly related to a going program in our country. It is particularly difficult for such a person to stay and get a Ph.D. For years there has been the attitude that people don't need as much training to work in a technically underdeveloped area as they do in a developed area. Consequently, persons have been given a series of short visits to laboratories in the United States for 6 to 12 months, and then have gone back and were expected to discharge creditably responsibilities which here we never dream of giving to an individual without 3 or 4 years of formal preparation or of supervised experience. There is beginning to be some realization on the part of certain foundations that people have to be well trained and also protected for a number of years. There is a real need for fellowships for training for long enough periods of time to acquire real professional competence in all of the basic sciences and the basic medical specialities.

PATWARDHAN: Our objective will be defeated if a plan for fellowship does not find some means of cutting through red tape and granting fellowships more quickly than has ordinarily been the experience in the past. Having to wait for years in some cases is a most disheartening experience for the fellows. The head of the institution cannot make plans with regard to the actual type of work that the fellow is going to do based on his training and experience.

STANBURY: Perhaps just to underline what Scrimshaw was saying, there is nothing more useless to us than a fellow who comes through and spends 2 or 3 days or even weeks as part of a junketeering trip through the States. I'd like to make a plea for these fellowships to be a minimum of a year so that the fellow can have a chance to settle down and absorb the technical information and the atmosphere of the laboratory.

MACKENZIE: I strongly urge that fellowships for individuals who have the equivalent of a Ph.D. degree and wish to come to this country and do post-doctorate work, be for 2 years. It is quite impossible for a man to come to a laboratory and start new research and achieve anything positive in less than 2 years. He is usually getting his first results somewhere around the end of the

first year. We also have to consider the laboratory that is playing host. If there is someone who is coming as a post-doctorate fellow who has an M.D. and no experience in research, he has to learn not only research techniques but research thinking. Two years is again a minimum and to have something useful come out, the fellow should spend 3 years.

KINNEY: This reasoning is pretty much in line with that of granting agencies now. They find that support for a project of any size for one year is almost worthless and that it should be given for a 2- or 3-year period.

PATWARDHAN: I am speaking now of the training of fellows who do not have a base to go back to after their training period is over. I have seen in this country in the last 2 months young men from my country who came here to spend 2 years, and who after completing post-doctoral fellowships have taken graduate fellowships. Now they want to go back to our country, but although we are supposed to be short of technical personnel, we somehow are unable to find jobs for them. The result is that some go to India, wait for 6 months to 2 years, and finally find jobs in disciplines where their training is completely wasted.

Another loss from our point of view is that these young men continue to stay in this country for 5 years or longer. Training programs will not be successful unless the person who is going to be trained has an opportunity to go back to his country and continue his work in the line in which he has been trained. The Rockefeller Foundation, I understand, has adopted this policy for India. It will take some time to know how it will pay off, but so far as my institution is concerned, it has paid off handsomely.

HIGGINSON: At the level at which such an exchange is most valuable, it often involves married men. We have found people that would be interested in exchange programs and the agencies are prepared to send them but they are not prepared to pay for wives and children, or else the individuals must lower their standard of living unduly.

PATWARDHAN: I heartily endorse the suggestion that there should be an exchange program at various levels for workers from one institution to another. This will provide experience in areas where ordinarily people have no opportunities to go. On the other hand, it will give workers from institutions in those areas who come to work in technically advanced countries a chance to study certain aspects of fundamental problems, for which they have neither the equipment nor funds at home. This will stimulate work on many things, but the difficulty pointed out by Higginson has to be borne in mind. I had, during my recent tour, occasion to discuss this subject at one or two universities, and I found that the subject always was introduced by the responsible persons in these places, and not by me. So I feel that there is a demand for an exchange of workers from one country to the other.

AYKROYD: By exchange, do we mean actual simultaneous exchange? Are we not thinking equally of the idea of sending one or two workers from this country to work for a year or so in some country where there are acute problems of nutrition, not necessarily on an exchange basis? Is that idea included in our present discussion?

SCRIMSHAW: A direct swap would hamper the system. We are confusing two things: first, training programs as such, where visits to an institution shouldn't be less than 6 months, and preferably should be a year. With exchange visits of highly competent people, in 5 or 10 days of round-table discussions a tremendous amount will be accomplished for both the visiting and host groups. We should provide for brief exchange visits, not necessarily on a simultaneous basis, of highly competent people, where it's almost a matter of consulting. Distinguish this sharply from exchanges for the purpose of training.

KINNEY: Next, I suggest we speak briefly of working conferences.

SCRIMSHAW: These have proved highly useful in the past. Such meetings as the two Macy conferences on protein malnutrition have done a tremendous amount to stimulate work in this field and to improve its quality. This conference on nutritional pathology will have a similar effect. We can safely go on record that carefully planned conferences with carefully selected participants can be extremely useful.

DARBY: I have something broader in mind. School teachers sometimes call it a workshop. I was thinking not of the present

type of conference which I call a talking conference, but one where people gather for a period of a month to two months, roll up their sleeves, and dig into the problem, looking at the pathology, getting into clinical studies in the hospital, or whatever opportunity offers. These would be trained individuals. Such an arrangement might prove exceedingly valuable; and I don't know of such opportunities being provided at present under any auspices.

SCRIMSHAW: One obvious practical way of doing this is for an institute to organize a conference around specific material that is available and invite people who have the training as well as experience with the clinical material. This, of course, is a matter of money being available for the organization of this particular type of workshop or working group, as well as for the talking type.

KINNEY: One variation of this would be a traveling workshop to provide for three or four men in different parts of the world who are concerned with the same disease to set up a working group which moves from area to area within a relatively short period of time. In this way they could review a large amount of material dealing with the problem on the spot and would be able to fit the picture together more clearly.

SCRIMSHAW: This would be ideal and perhaps necessary for certain subjects.

RAMALINGASWAMI: I want to describe our experience with working groups. Twice a year, seven or eight persons meet in the offices of the Indian Council of Medical Research and bring whatever material they have on liver disease which they have collected experimentally or in the course of their hospital work. This material is seen by all the persons around the table. Each person makes his own report and there's a final discussion. Also one or two subjects are set apart for special discussions. We found these to be very workable and extremely valuable meetings because, India being a large country, there is a lot of variation in the type of picture that one sees.

NADEL: In addition to the working conferences that have been described, I would like to bring to your attention the one-man conference type that recently took place when Paul Steiner visited various centers in Africa. This is an approach which provides a means for a competent man to take a uniform look at all the material of a type of disease. It would be possible, for example, for one man to study the variety of unusual cardiac lesions that occur in different parts of the world in consultation with local pathologists or clinical nutritionists.

KINNEY: Does this group feel that there are other nutritional deficiency diseases or diseases influenced by dietary deficiencies on which a conference of this type could be profitable within the near future?

DARBY: I certainly feel that there are such needs. For example, WHO is holding a study group on nutritional anemias in September. The subjects of pellagra, neurological consequences of diet, urinary calculus, protein malnutrition and unusual types of heart disease have already been mentioned as topics in which there's need for clarification. There must surely be many other subjects which will come up from time to time. Also, a single item such as beriberi will need more exhaustive treatment at some future date when additional data accumulate, much as has been done for kwashiorkor. I think it would be important to go on record as favoring such conferences.

KINNEY: Nothing has been said about the availability of grants from the Public Health Service to support research in areas outside of this country. I believe it would be worthwhile to have some statement of official policy on this. Nadel?

NADEL: I can read you a policy statement published in October 1956. "Grants may be made in support of research work conducted outside the territorial limits of the United States under the auspices of foreign organizations only when satisfactory evidence is provided that the research is of such nature that for scientific reasons it can best be conducted outside the United States and is of general importance to the health and medical sciences."

KINNEY: From what we have heard concerning the needs for investigation of the diseases taken up during the past three days, I would certainly think that research grant applications from foreign laboratories would be welcomed in order to help solve some of the problems which we have discussed and which can only be investigated where such diseases are occurring.

What is the present status of support for training centers outside of this country?

HUNDLEY: The National Institutes of Health have recently put quite a bit of support into training centers in the U.S. This undoubtedly will be useful but will not meet all the needs, but certainly there is some need for some strengthening of such training centers in other countries. The extent to which this could be implemented through the National Institutes of Health program is not known to me.

SCRIMSHAW: The NIH grants which have been given outside the country, and I can speak particularly of the one given to INCAP, have been of tremendous help in making possible the training of local people in programs which would have never been possible otherwise. We have used the funds which supported the protein malnutrition work to bring in a series of Central American physicians and biochemists, and from these men we have been able to select those who could be sent for further training. There isn't any question that from this one grant alone will come several people who will be making permanent contributions to the field of nutrition. It is inevitable that National Institutes of Health grants will have this very beneficial indirect effect of training individuals as well as whatever direct effect comes out of the research itself.

RAMALINGASWAMI: I wanted to support the idea that something should come out of this conference bearing on pathology and underscoring the importance of pathologists looking more and more into nutritional disease, because both pathologists and pathology will gain by a study of human disease. Most of the contributions have been on experimental pathology in animals. I think this conference should bring out the need for studies on the pathology of human nutritional disease.

RECOMMENDATIONS OF THE CONFERENCE

The members of the conference voted to go on record as favoring the following recommendations which they feel will improve the status of research and training in nutritional disease. These recommendations were formulated during the course of considerable discussion by the participants and are presented in this form for the sake of clarity.

I. It is recommended that a mechanism be set up to provide means for scientists at appropriate levels of training to study nutritional disease throughout the world. Consideration should be given to provide fellowships for scientists at all levels to study nutritional problems in endemic areas.

Progress in the study of nutritional disease will be accelerated if financial support is provided for the exchange of professional staff between institutions in technically advanced countries and countries with endemic nutritional and related diseases of unique interest.

II. There is a need for conferences on nutrition of two basic types, i.e. working conferences and review or evaluation conferences. Both types should receive strong financial support.

a) The working type of conference in general, would be composed of a few highly qualified investigators who would be convened to work out technical problems associated with specific nutritional disease. The working conference also would be of value in reference to diseases and syndromes thought to be dietary in origin where there are substantial and unresolved differences in regard to their identification. In such circumstances there is a need for small teams of specially competent workers to visit endemic areas in order to attempt to resolve such differences.

b) The review or evaluation type of conference was considered to be helpful in delineating the needs for research in areas of nutritional disease. In reference to the three disease entities under discussion the conference hoped that sufficient progress would be made to justify having a similar conference in 3 years.

III. Support should be provided for scientists from less developed areas to receive training at all levels in countries that are technically more advanced. At the present time, although there are a number of sources of support for these individuals, relatively few fellowships provide for training in the basic sciences. Support for the above purposes should be for at least 1 and

preferably 2 or 3 years in duration. Preference in the awarding of such fellowships should be given to those individuals who have a firm commitment to return to a post where they will be supported by their institution in an appropriate manner.

IV. The Conference noted that the National Institutes of Health had provided substantial funds to increase training opportunities for scientists interested in nutrition at several centers in the United States. All indications point to the success of this program. The Conference felt there is an urgent need for additional support for centers outside the United States where nutritional conditions of particular interest exist which provide unique and needed research and training opportunities.

V. It is recommended that training programs in nutrition for medical students, graduate students in the preclinical sciences, and public health workers of all types, be augmented and improved.

VI. The Conference recognized the contribution that pathology and its research techniques have made to the understanding of nutritional disease. It pointed out that a world-wide shortage of well-trained experimental pathologists exists. It urges that support be provided for the training of pathologists so they will secure a broad biological and medical background which will serve to orient their research to the pathogenesis of disease. It is hoped that such trained experimental pathologists will be attracted to the field of nutritional disease.

VII. There is a need for an increased awareness by nutritionists, biochemists and other scientists working in the field of nutrition of the necessity for a pathologic interpretation of their material, if such material is to be utilized to the fullest. This need often can best be met by a collaboration of such scientists with pathologists interested in nutritional diseases.

VIII. It was recognized that there was need for a mechanism for the exchange of pathologic material. It was suggested that support be provided for a center or centers which would facilitate the exchange of material between scientists, but would not itself serve as a storehouse of material.

IX. It was noted that there is a pressing need for clinicians and pathologists to standardize criteria for the diagnosis of nutritional deficiency diseases.

X. It was the consensus of the Conference that the discussion had pointed up the need for additional information regarding the pathogenesis, as well as the incidence and severity of vitamin A deficiency, goiter and beriberi. By inference, this need could be extended to other deficiency diseases. It was recommended that investigations be expanded and supported by research grants.